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VOL. II.—41ST YEAR SYDNEY, SATURDAY, NOVEMBER 13, 1954 No. 20

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THE GENERAL PRACTITIONER AND PUBLIC HEALTH ADMINISTRATION.[1]

By C. E. COOK,

Canberra.

THE purpose of my talk today is twofold: first, to invite your attention to the importance, indeed the indispensability, of the general practitioner in any organized system of preventive medicine; and secondly, to prompt you to initiate a movement within the profession to have the family doctor integrated into your own State's public health organization. I wish to impress upon you that I shall speak, not as an officer of the Commonwealth Department of Health expressing the views of the Department whether avowed or supposed, but as a fellow member of the British Medical Association, and one of a family in which all the bread-winners of three generations in Australia have been medical practitioners.

Let me first state the functions of a public health department as I see them. These I believe to be the following: (*a*) to study the incidence of disease and the causes of morbidity and mortality in the community; (*b*) to identify the factors which are preventible and to devote itself to removing them; (*c*) to initiate and foster research into improved methods of diagnosis and into methods of treatment calculated to shorten the period of illness or avert a fatal outcome, to extend the knowledge of these, and to provide facilities for their universal application.

Public health departments as they exist today are successors to the English organizations of a century ago, and in structure, function and technique have not materially changed since first designed. The sudden population movements of the industrial revolution produced in Britain conditions of extreme overcrowding and squalor. These in turn were associated with an appalling incidence of and mortality from infectious disease. The relation of these phenomena as cause and effect began first to be realized about the middle of last century, when Chadwick (1842) reported upon the sanitary condition of the labouring population of Great Britain. He ascribed epidemics to filth, overcrowding and impure domestic water, and advocated for their control organized drainage and scavenging, improved water supplies, and better ventilation and lighting in dwellings. His "sanitary idea" hypothecated that all epidemic disease was produced and fostered by the effluvia and miasmata associated with bad environmental sanitation. Completely unaware of the infectious nature of communicable disease and the part played by environment in assisting the passage of infection, he eventually succeeded in stirring a national effort to improve the sanitation of towns as a means of preventing disease. Having regard to his premises, it was natural, indeed

[1] Read at a meeting of the Federation of Country Local Associations, New South Wales Branch, British Medical Association, Canberra, April 20, 1954.

inevitable, that when his recommendations were implemented the functions and duties of public health administration were vested in the local authority, and that the enabling legislation was restricted in its scope to activities within the capacity of that body to perform. With the local authority powers under the *Health Act* have remained ever since, subject in Australia only here and there to advice from a trained medical officer of health. In this country subsequent amendments to the original legislation under which the local health authority operates have elaborated the old rather than introduced new functions; indeed, what expectation could there be that such a body would either initiate a demand for wider powers in preventive medicine or attain the technical competence to warrant being entrusted with them?

The improvement in environmental sanitation achieved by these bodies has resulted in the elimination of a great mass of disease, which the old conditions directly fostered; but the disappearance of these has revealed a substratum of preventible disease which is not susceptible of further reduction by the measures available to a lay operative staff. It might be said that we have in great measure corrected the epidemiological factors introduced by our habitual concentration in fixed communities, but have still to eliminate those which would confront an uncivilized nomadic people. Nevertheless, the organization of the public health departments in most Australian States has not been modified to meet the new situation.

Let us consider briefly a few of the ingredients of that persisting hard core of disease which cannot be expected to yield to Chadwick's methods, however zealously and efficiently applied.

Acute Rheumatism.

Precise information regarding the incidence of acute rheumatism in Australia is unobtainable. That the disease is only too common, however, is revealed by the fact that amongst the infectious diseases rheumatic heart disease is the most consistently high killer in the Commonwealth except tuberculosis. Bryan Dowd says of New South Wales that rheumatic fever is the commonest cause of death in the ten to fourteen years age group, and that one-fifth of the medical beds in the Royal Alexandra Hospital for Children are permanently occupied by patients suffering from rheumatic heart disease. Indeed, if allowance is made for medical beds devoted permanently to the care of infants requiring the correction of feeding errors, the proportion allocated to rheumatism becomes two-sevenths.

Some idea of the incidence of rheumatism in Australia may be gleaned from the experience of the United States of America, where it is estimated that 2% of school children suffer from rheumatic conditions of the heart, and that rheumatic fever is responsible for 80% to 90% of all heart disease in persons aged below twenty years. Three hundred thousand persons have been rendered unemployable by rheumatism, and 147,000 are permanent invalids.

Attempts to assess the incidence of rheumatism in Australia have not so far been successful. Only recently have certain States been induced to make the acute condition notifiable. Medical practitioners have failed to cooperate in notification for a variety of reasons, and the departments, always reluctant to increase the list of notifiable diseases, have contemplated discontinuance of notification rather than recourse to measures designed to improve its efficiency. Meantime, the organization for public health administration being what it is, medical practitioners who have notified rheumatic fever have been dumbfounded and embarrassed by the agents of local authorities visiting the homes of the patients and insisting upon house disinfection.

Salmonella Infection.

Throughout the year in all the Australian States, epidemics of food poisoning are numerous, recurrent and extensive. Although Salmonella infections are now notifiable in three States, notification, depending as it does upon bacteriological identification of the causal organism, represents but an infinitesimal fraction of the amount of disease actually occurring. Some idea of the prevalence of these infections may be derived from the number of prescriptions under the *Pharmaceutical Benefits Act* for drugs used exclusively for the treatment of diarrhœa and dysentery —sulphaguanidine, phthalylsulphathiazole and succinylsulphathiazole. In 1953 these totalled 168,000. As a measure of the incidence of Salmonella infection, this figure must be recognized as excluding sufferers treated as either in-patients or out-patients by hospitals, and patients treated with antibiotics or by more traditional methods or not treated at all.

These infections being due to the unclean preparation of food in the hands of the individual or to the use of food from infected animals, control depends upon identifying the particular organism involved and tracing the source of contamination. For this task the local authority is unequipped, and only the general practitioner in contact with the patient can provide the basic information and material to form the starting point for investigation.

As an indication of the importance attaching to a thorough study of epidemics caused by Salmonellæ, apart from the objective of controlling them themselves, let me recall to you a recent experience in the eastern States. For several years typhoid fever has occurred only sporadically and is now but rarely seen by the general practitioner. Throughout New South Wales cases have averaged only about one per month in a normal year. Towards the end of June and early in July, 1953, an unusual prevalence of typhoid was noticed in New South Wales and Victoria. By the end of July notification in all States totalled 12 for one week. During the next seven weeks there were 101 cases, and for the six months ending December 31, 1953, the total was 175. Infection was traced to contaminated imported food; but the point of crucial importance was that the clue permitting the source to be identified was the association of *S. typhi* with a Salmonella new to Australian pathology—*S. senftenberg*. It was subsequently ascertained by bacteriological investigation that *S. senftenberg* had been a contaminating organism in this product for many months previously, doubtless in that time contributing significantly to the incidence of food poisoning. Had the notification and identification of Salmonella infections been efficient, the contaminated character of the imported food would have been recognized months before it was, and effectual action then to control the sale of the product would have prevented the outbreak of typhoid without a case occurring.

Influenza.

The possibility of a virulent pandemic of influenza of the character of that of 1919 must always be faced, and it is the responsibility of health departments to do everything possible to avert this. Control by vaccine can be quite efficient, but it depends upon isolation of the specific strain of virus involved. At least five weeks under optimal conditions is required to complete a batch of influenza vaccine. It is obvious, therefore, that if an epidemic or pandemic is to be averted by mass vaccination, public health practice must permit the early recognition and prompt typing of the virus involved, and a full and free interchange of epidemiological information between States and countries.

An organization capable of giving this service can be based only upon full and trained cooperation at the general practitioner level, since it involves not only notification of suspected disease, but the prompt collection and transmission of virological material.

Virus Pneumonia.

Primary atypical pneumonia or virus pneumonia was certified as the cause of 71 deaths in 1952. The number of fatalities may have been considerably higher, for of 2863 deaths attributed to pneumonia the type was unspecified in 609, and 1505 were simply described as bronchopneumonia.

Contributing to the incidence of this condition are influenza, psittacosis and "Q" fever. Psittacosis is probably much more prevalent in Australia than has been suspected. Although it is notifiable, total notifications are not more than one-half the proven cases. Miles is of the opinion

that the high mortality reported in Australia is probably due to failure to recognize or identify mild cases. Recently an epidemic of aseptic meningitis at Port Augusta, in the course of which there were some 30 cases, was found to be caused by a virus similar to that causing psittacosis. Miles reports that 3% to 4% of inland aborigines examined in the Northern Territory had antibodies to psittacosis in their serum. It is important that the true incidence of psittacosis be ascertained; but obviously this can be effected only with full cooperation of the medical attendants in charge of patients. It is important because psittacosis contributes in an unknown degree to the incidence of virus pneumonia, and because the broad spectrum antibiotics used in the treatment of virus pneumonia increase the carrier rate of psittacosis infections. Miles is of opinion that a quite small variation in the character of the virus would give psittacosis a much greater public health significance than it is accorded today, and an efficient public health department should be alert to anticipate and forestall such a contingency.

The incidence of "Q" fever in Australia has not been carefully ascertained. This infection also contributes to the morbidity and mortality associated with virus pneumonia. Determination of its incidence, relying as it does upon serological examination of the blood of patients and convalescents, is essentially a matter for initiation by the general practitioner.

Cancer and Heart Disease.

You may hold that the general practitioner can contribute little or nothing to the solution of the problems presented by cancer and heart disease, those two major killers. I suggest, however, that for statistical studies of early signs or for late review none can better provide the fundamental information necessary to complete study. None, moreover, can better assess the need for local aids to early diagnosis or supply information upon requirements for early and effectual treatment.

Infant Mortality.

For every 12 surviving live births in Australia one infant life is lost by stillbirth, prematurity or birth trauma before the end of the first week of extrauterine life. Action to identify and correct the factors principally contributing to fœtal death, prematurity and neonatal mortality in this country must await the laborious collection of a mass of statistical data. The National Health and Medical Research Council has recommended adoption throughout Australia of a method of collecting this information based upon a medical certificate of birth and a special certificate of fœtal death. Although medical practitioners chiefly concerned—the Royal College of Obstetricians and Gynæcologists—have in all States endorsed and strongly supported the recommendation, progress has been halted at the administrative level because statisticians, registrars-general and health departments, who must together provide the administrative machinery for collecting and collating the information, have hesitated to ask for the necessary cooperation of the individual practitioner. I am persuaded that the profession as a body would regard collection and collation of this information as a most important forward step, and that it would welcome the opportunity to cooperate, but because there are no liaison between department and practitioner, no means of explaining the method and purpose to the profession, and no opportunity to ascertain the practitioner's point of view, we have reached a condition of stalemate. In this as in all else it is quite clear that there is a compelling need for some vocal and executive organization of the general practitioner in public health.

If you recall for a moment the functions of a modern health department as set out earlier in this talk and relate them to the control of the conditions since discussed, I am sure you will be impressed by two facts: (i) There could be no prospect of reducing the incidence of these afflictions by any means available to a lay authority or by the most zealous application of conventional local health authority methods. (ii) To have any prospect of ascertaining the incidence of or reducing the morbidity and mortality from these conditions, a department of public health would require some effectual power of direction over the general practitioner to secure his active and complete cooperation in the diagnosis and reporting of disease and in the application of prophylatic and therapeutic measures designed for its control.

In addition to those incidental to its interest in specific diseases, a health authority in pursuit of the objectives cited may be expected to make other calls upon the cooperation of the general practitioner.

Health Education.

Effectual and complete control of preventible disease is practicable only in a community so enlightened and disciplined that the individual without conscious effort himself on all occasions applies the controlling measures and habitually conforms to the sanitary prohibitions dictated by prophylaxis.

A modern and well-concerted plan to prevent disease must include the education of the mass of the people in prophylactic measures which are the responsibility of the individual, and the inculcation of a self-imposed discipline which will assure their universal and constant application.

Of all persons concerned with the study, prevention and control of disease, who is better situated to undertake this task than the family doctor, who sees the individual in his own environment, is trained to perceive the dangers of habits and practices contributing to disease, and can with assurance warn and advise upon their correction?

Immunization.

The public health department may aspire not only to the universal artificial immunization of the child population against certain diseases, but to the preservation of accurate, accessible and permanent records of inoculations performed. These could be readily obtained in organized public prophylactic clinics; but haphazard and unrecorded immunizations in private practice may prove an intolerable embarrassment.

Child Health Services.

Most health departments have come to realize that there are many inadequacies and imperfections in the medical and health services provided for the infant and for the pre-school and school child, and that these can be remedied only by invoking the more active cooperation of the family doctor in the work of the service. Calls upon the general practitioner for these services may be more exacting in one State or locality than in another; but any departmental response to the current impulse towards improving these services must involve the general practitioner either in closer liaison or in conflict with the health authority.

Use of Antibiotics.

The widespread use of antibiotics presents several problems. The broad spectrum antibiotics offer the busy practitioner an opportunity to effect the prompt cure of many infectious diseases without the delay, trouble and expense of making a precise diagnosis. The health authority cannot afford to overlook the following serious consequences of this practice: (*a*) The practitioner's aptitude for clinical discernment and appreciation is apt to be impaired by disuse. (*b*) Inappropriate use or dosage may promote the development of resistant strains amongst organisms at present susceptible. (*c*) Patients may be rendered sensitive to the antibiotic at a time when its use is not required, and thereby denied it when its exhibition may decide between life and death. (*d*) The patient may be converted to a healthy carrier of infection undiagnosed and unsuspected, or he may, by a disturbance of a biological equilibrium in the bacteriological flora carried, be left vulnerable to another and possibly more serious infection. (*e*) A costly therapeutic agent purchased by the Treasury may be used unnecessarily instead of a cheap and equally or more efficient alternative.

Psychosomatic Medicine.

Amongst the morbid conditions contributing heavily to the sum of human misery and the aggregate cost of medical treatment, hypertension, peptic ulcer, rheumatoid arthritis and others are now claimed to have an emotional background, recognition and palliation of which may promise real relief. Every actively functioning and progressive department of health will be impatient to take up the challenge of this doctrine, to assess its verities and to exploit its realities in the prevention of suffering and the reduction of therapeutic costs. For these purposes, what agency could it use more conveniently situated or more suitably trained than the family doctor—indeed, what other agency exists?

Discussion.

One could multiply these instances almost to the exhaustion of all clinical conditions known to medicine; but I hope I have cited sufficient to prompt in you a recognition that the time has arrived when new measures for the study of disease and its prevention are an inescapable necessity. I hope, too, that I have convinced you that the proper functions of a health department involve, under modern conditions, a definite integration of the practising profession into any system of organized preventive medicine, and a strict discipline of its individuals either voluntarily self-assumed or statutorily imposed.

You may be prepared to acknowledge the principle that the general practitioner must play an important, indeed a decisive, part in any improved organization for the prevention of disease; but you may feel that the calls upon your time in practice preclude your undertaking it. You will argue, perhaps, that the responsibility of resolving this deadlock must rest elsewhere, and that the initiative lies not with you but with the department set up and maintained by government to devise and apply measures for improving the public health.

Let me remind those of you who share this last conviction that most of the health departments in Australia have been developed in the Chadwick tradition. Their structure and the legal codes which they administer are outmoded. The limited scope of the State Health Acts and their dependence upon local application by lay administrative bodies have together operated to frustrate the expansion of health departments into new fields.

Governments have been understandably reluctant to impose new tasks involving medical or technical knowledge upon local health authorities, and problems created by the inertia or incapacity of these to cope with new specialist functions have usually been met by the creation of new segregated departments operating under special laws. Hospital management, for example, has been divorced from health administration in some States, and from the control of poisons and therapeutic substances in most. Health departments in this way have been deprived of sources of information and denied opportunities for intervention which are fundamentally essential to the proper discharge of their own functions.

Adoption of a modern and improved pattern of health administration will involve in some States reorganization of the health department, not only to permit its assumption of functions and duties proper to its purpose which are at present excluded from its scope, but also to restore it to the administrative status from which, over the years, it has been deposed.

The magnitude of this task with its calculated assault upon vested public service interests makes it one unlikely to be undertaken by any State Government on the initiative of an unvalued and submerged health department or in the absence of an insistent public demand.

That public agitation for a comprehensive revision of health administration must develop I shall now endeavour to show. Let me, however, preface my remarks by stating my conviction that unless this agitation is deliberately canalized to these ends, it will not be directed either toward State Governments or toward improved preventive medicine.

In this country the incidence of taxation for social services is not light, and it extends to the great mass of wage-earners. The financial stimulus which first demanded government assistance in lightening the burden of medical expenses incurred by the individual must, as he gradually becomes aware of his continuing, if less manifest, obligation, be redirected towards a demand for reduction in costs.

Under a national health service scheme, unless special corrective action is promptly taken, costs must be expected to rise progressively for many reasons. To quote but two, one may mention the impulse of the individual to obtain each year full value for his annual tax contributions, impelling him to seek attention for trifling conditions which otherwise he would ignore, and the tendency of the medical profession to have recourse to costly methods of diagnosis and treatment formerly avoided out of consideration for the financial condition of the patient.

Costs already high are considerably inflated by several of the conditions we have considered as possibly being preventible. During 1953, for example, the cost of prescriptions for drugs used exclusively for the treatment of enteritis outside hospital was over £100,000—just one-sixtieth of the total Commonwealth expenditure on pharmaceutical benefits for the year 1952–1953. This figure, be it clearly understood, takes no account of the expenses incurred by the Commonwealth in the treatment of diarrhœal disease by antibiotics or by sulphonamides other than those mentioned, nor does it include the cost of treatment of in-patients or out-patients by hospitals. The cost of rheumatism to Sydney's Royal Alexandra Hospital for Children alone approximates £40,000 annually. The aggregate disbursement by the community for its treatment in other hospitals and in private practice, or involved in the payment of pensions, insurance claims and the like, is incalculable.

It is an anomaly of our federal system that the authority collecting the social services contribution and providing funds for the treatment of disease under the *National Health Service Act*—the Commonwealth—has to hand no direct means of developing a system of preventive medicine with the object of reducing the costs entailed in the treatment of preventible disease. By contrast, the authority charged with the prevention of disease—the State health department—incurs no comparable financial disadvantage from its failures nor commensurate advantage from its successes.

Increasing agitation to reduce the social services contribution without impairing benefits is likely therefore to be an embarrassment only to the Federal authority and to leave the State unperturbed. Any response to a public demand for economy must be expected first in the field of Commonwealth administration. Faced with a necessity for economizing, the Commonwealth is likely to turn first to revision of the fees paid for therapeutic services—a probability which gives you individually and collectively a financial interest in the initiation and furtherance of plans to improve and extend measures for the reduction of morbidity throughout Australia.

As an alternative, or as a supplement to revision of the fees paid for service, the Commonwealth can have recourse to some device whereby acceptance of a therapeutic fee by a medical practitioner will involve him in an obligation to perform one or more routine services required as part of a concerted national research or control project. Such a device might well, unless carefully planned and policed, sooner or later deteriorate into a form of regimentation repugnant to you.

Conclusion.

In closing, let me suggest that you have a national obligation, a financial incentive and a professional interest in the reformation and reactivation of public health administration in Australia, in order that there may be

evolved a system of preventive medicine better designed to cope with the new problems revealed by present knowledge and more readily able to grasp the opportunities offered by modern research. Who, better than yourselves, can provide the central health authority, whether State or Commonwealth, with the active inspiration derived from daily contact with the personal problems of the patient and with the realities of medical practice?

Let me express the hope that you will promptly set yourselves to planning and to discussing with your State health department the means by which your voluntary services may be most usefully mobilized and amicably concerted in an enlightened and intelligently designed routine for the reduction of human suffering and for the most profitable use of available financial resources. Let me reaffirm my belief that you have, in addition to a moral obligation, a material interest to do so, more particularly as the hour is already late.

THE EFFECT OF THE INTRAVENOUS ADMINISTRATION OF SODIUM THIOSULPHATE ON BLOOD LEAD CONTENT AND ON THE EXCRETION OF LEAD IN URINE AND FÆCES IN CASES OF LEAD POISONING.

By D. O. SHIELS, W. C. THOMAS, G. R. PALMER, P. CORNISH and E. KEARLEY,
Industrial Hygiene Division, Department of Health, Melbourne.

THE value of sodium thiosulphate when used intravenously in the treatment of acute and chronic lead poisoning (although not the sequelæ) has been amply demonstrated (Dennie and McBride, 1923, 1924; Ockerblad, 1923; Pincus, 1936; Schmitt and Lossie, 1938, 1939; Shiels, 1952).

The mode of action of sodium thiosulphate has, however, not yet been clearly demonstrated. Dennie and McBride (1924) at first suggested that lead was precipitated as sulphide and excreted as such, but gave up this view in favour of one according to which the metals (arsenic, bismuth, mercury and lead) were removed from combination with proteins by sodium thiosulphate and rendered soluble. They further stated that sodium thiosulphate could be used as a diagnostic aid in suspected cases of poisoning by these metals in which no metal had been found in the urine previous to such use, as it would increase the elimination of the metals.

Schmitt and Lossie (1938, 1939) stated that in one case of lead poisoning treated intravenously with sodium thiosulphate the blood lead content was significantly decreased after eight days and returned to normal after fourteen days, and that in cases in which an increase in blood lead content was due to cirrhosis of the liver, sodium thiosulphate lowered the blood lead content and eliminated lead from the depots.

The originals of these last-mentioned journals are not available in Australia, so the actual quantitative results are not known.

PRESENT INVESTIGATION.

At Mount Isa, where this method of treatment had become the routine, it was observed that remissions after adequate treatment with thiosulphate were very rare, and further, that the lead line disappeared rapidly during this treatment.

It was considered important to know the effect of the treatment on the freely circulating lead in the body, whether it caused storage in a less soluble form in the bones, or whether it increased elimination in urine and/or fæces.

Earlier Work: Effect of Thiosulphate on Concentration of Lead in the Urine as Determined by Modified Taylor Method.

The dose was 30 grains in a few cubic centimetres of water given intravenously every second day.

The effect of the thiosulphate was to cause a rapid decrease in the urinary lead concentration as determined by Taylor's method (1925) of precipitation with, or adsorption on, calcium oxalate, ashing of the precipitate in the dry way and subsequent comparison of turbidity produced in the final lead solution by sodium bisulphite with standard solutions of lead. A slight modification (Shiels, 1936) of the method was employed in which silica dishes were used instead of platinum, and nitric acid instead of hydrochloric acid for the solution of the original precipitate prior to ashing.

TABLE I.

Subjects.	Urinary Lead Concentrations.				
	Mean Values (Milligrammes per Litre).			Percentage Decrease on Initial Value.	Value at 8 to 10 Days as Percentage of Initial Value.
	Initial.	At 8 to 10 Days or Less.	Decrease.		
Group A: 14 subjects treated with thiosulphate	0·321	0·128	0·193	62·8	37·2
Group B: 4 subjects not so treated ..	0·274	0·211	0·063	21·47	78·53

Since these modifications did not affect the original step of recovering the lead from the urine or the final determination by sodium bisulphite, the method will be referred to as the modified Taylor's method.

Table I shows in summarized form the effects on the concentration of lead in the urine of 14 persons with lead poisoning all undergoing thiosulphate treatment (Group A) and of four persons in Melbourne not having this treatment (Group B), and Table II the results of statistical analysis of the findings.

TABLE II.

Decrease in Urinary Lead Concentration as Determined by Taylor's Method. Significance of Mean Decrease.

Observation.	Group A.	Group B.
Mean decrease at 8 to 10 days	0·193 milligramme per litre.	0·06 milligramme per litre.
Standard error	0·03259.	0·0216.
n	13 (14 subjects)	3 (4 subjects).
t	5·92.	2·88.
P	0·001.	Between 0·05 and 0·10.
Meaning of *P*	Highly significant.	Not significant.
Mean decrease at 8 to 10 days as percentage of original values ..	62·8.	21·48.
Standard error	6·114.	7·149.
n	13 (14 subjects).	3 (4 subjects).
t	10·27.	3·000.
P	0·001.	Between 0·05 and 0·10.
Meaning of *P*	Highly significant.	Not significant.

The results shown in Table I were determined by the modified Taylor's method.

There was no significant difference in the mean concentration of lead in urine for the two groups A and B prior to the commencement of the thiosulphate treatment, the values being 0·321 and 0·274 milligramme per litre respectively, or if one very high value—namely, 0·80—is omitted, 0·284 and 0·274 milligramme per litre respectively.

There was a rapid fall in urinary lead concentration for all those undergoing thiosulphate treatment except in one case, and no comparable fall in the case of those not on this treatment.

The fall in urinary lead concentration which took place during the thiosulphate therapy was statistically highly significant, and not due merely to daily variations which are known to occur in the lead excretion.

No such significant fall occurred in the case of the subjects who were not having thiosulphate injections, but who had ceased to be exposed to the lead hazard which had produced their condition.

For purposes of comparison the urinary lead concentrations at eight to ten days or less after cessation of exposure have been used. When an actual determination was not made at this time (eight to ten days) or just prior to it, the probable value has been estimated from the value at a later time, a steady fall of concentration with time being assumed.

Owing to the shape of the usual curves for concentration against time, in which there is a more rapid fall in the early days of treatment than in the later, this procedure would tend to favour group B, if anything, since an interpolated value at ten days in the case of the group A persons would probably be higher than the actual value at that time.

Since there was a rapid fall in the concentration of lead in the urine, and no compensatory increase in the volume passed, it appeared, if thiosulphate exerted its beneficial action by increasing the elimination of lead, that it did so via the fæces.

There were other possibilities.

Certain in-vitro experiments had indicated that in the presence of sodium thiosulphate aqueous solutions of lead acetate containing the lead and the thiosulphate in about the proportions which might be expected in the blood of lead-poisoning subjects, when treated with thiosulphate, failed to give the usual reactions of inorganic lead ion. It was thought, therefore, that the action of the thiosulphate might be to combine the lead or a considerable proportion of it in a complex negative ion, in which the lead was less active physiologically than in its normal occurrence, and that this lead was not completely precipitated with the calcium oxalate, or adsorbed on it, and that the results obtained by the modified Taylor's method were lower than the real values.

Another possibility was that precipitation or adsorption was complete, but that lead was subsequently lost in the various manipulations.

The addition of thiosulphate to specimens of urine containing known amounts of lead made no significant difference in the values found by the modified Taylor's method.

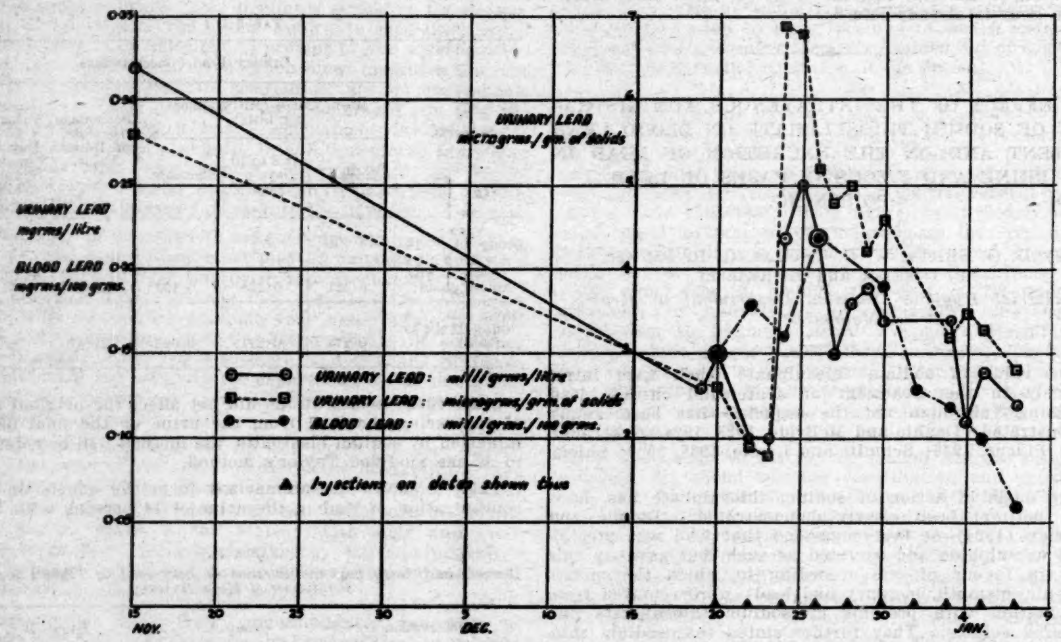

FIGURE I.
Subject number 14.

Unpublished work carried out in 1946 indicated that the Taylor method gave low values for the urinary lead concentrations when the subject was having thiosulphate treatment, and this has been confirmed by later work.

Effect on Urinary Lead Concentration as Determined by Wet Oxidation Method. Comparison of the Results by the Two Methods.

Recently a considerable number of determinations of urinary lead concentration for the urine of subjects not receiving thiosulphate treatment and of those having this treatment have been made, both by the modified Taylor method and by a wet oxidation method in which oxidation by nitric acid or nitrosyl sulphuric acid was followed by determination of lead concentration by the mixed colour dithizone method.

Statistical analysis of the results by the two methods has given the following findings.

There is no significant difference in the results of analysis for urinary lead content by the two methods for urine of persons not receiving thiosulphate treatment.

There is a very significant difference in the results for urinary lead concentrations determined by the two methods

for urine of persons who are receiving thiosulphate treatment. The values obtained by the wet oxidation methods were about 60% higher than the others.

Further, in the case of persons who are receiving thiosulphate treatment, the differences between the values as determined by the two methods are greater for urine collected during the twenty-four hours subsequent to the injections than they are for urine collected during the twenty-four hours prior to the injections. This will be referred to later, when the detailed results of subject 15 are being considered.

The results on which this statistical analysis was made are summarized in Table III.

TABLE III.
Concentration of Lead in Urine.

Specimens of Urine.	Mean Values (Milligrammes per Litre).	
	By Modified Taylor Method.	By Wet Oxidation Method.
25 from large number of persons not receiving thiosulphate therapy ..	0·165	0·175
25 from four subjects undergoing thiosulphate therapy	0·164	0·268

Detailed Consideration of Cases.

Subject 14 had been engaged on spraying a lead-containing enamel or glaze onto ironware (baths *et cetera*) and had an attack of colic in March, 1950, and was off work for a week, but was not diagnosed as suffering from lead poisoning. In August, 1950, he had another attack of colic and he was admitted to hospital on August 12. He was thought to be suffering from intestinal obstruction, and laparotomy was performed. He was discharged on August 17 and returned to work on August 31. He was readmitted to hospital on November 14, 1950, and recognized as suffering from lead colic. He was treated with calcium salt given by mouth and intravenously, and was discharged from hospital on November 20. He returned to work on November 26, but not to his previous hazard. He had another attack of colic on December 16, 1950, was readmitted to hospital on December 18, and then treatment with thiosulphate was instituted on December 20. He had a course of 10 injections and was discharged from hospital on January 8, 1951. He had no recurrence of symptoms.

The results of the determinations of the lead content in blood, urine and fæces and the total excretion of lead for this subject are shown in Figures I and II. Injections were given at about 10 a.m., but the collection of urine covered the periods from about 9 a.m. daily, so that there was some overlapping.

In this case of a person who had been away from a significant lead hazard for thirty-four days prior to commencement of thiosulphate treatment, the effect was to cause a substantial rise in the true urinary lead concentration and total daily urinary lead excretion after the third injection, which was maintained for about twelve days. The results for the lead content of blood and fæces will be referred to later.

Subject 15 had been employed in a factory making electrical accumulators, and was admitted to hospital on June 4, 1951, with abdominal colic. He had lost weight. On June 8, examination of his blood revealed 20,200 stippled cells (including 1800 coarsely stippled, 2400 moderately stippled, and 16,000 finely stippled cells) per 1,000,000 red cells. The ratio of monocytes *plus* large lymphocytes to small lymphocytes was 0·83. Calcium gluconate given intravenously controlled the colic. The initial urinary lead concentration was high—0·83 milligramme per litre. On the fourth day in hospital sodium thiosulphate injections were commenced. They were given on alternate days at 12 noon. Urine was collected (in specially prepared bottles containing a few cubic centimetres of redistilled chloroform) during the twenty-four hours subsequent to the injection, and separately for the next twenty-four hours—that is, for the twenty-four hours prior to the next injection. Fæcal samples were collected similarly during the twenty-four hours subsequent to each injection and for the twenty-four hours prior to the next, the times of defæcation being noted.

Blood samples were collected at the time of injection.

The data in relation to the urine, blood and fæces are shown in Tables IV and V and in Figures III and IV.

Improvement in clinical condition was rapid, and the patient was discharged to the out-patient department on June 25. His subsequent treatment was by sodium citrate, but this period is not considered in this paper.

The effect of the thiosulphate treatment in causing differences in the urinary lead concentrations as determined by the two methods, which has already been referred to, is also well shown by reference to the results for

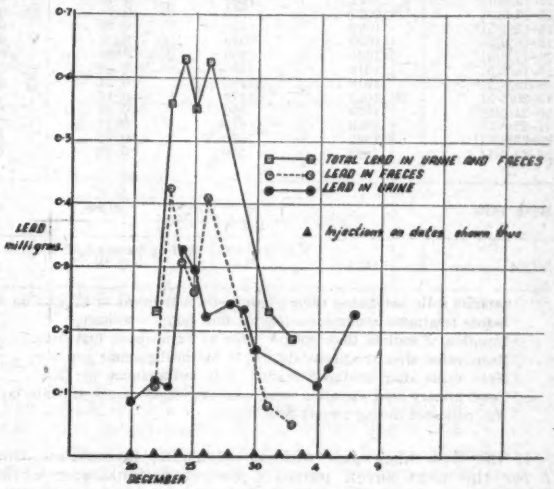

FIGURE II.
Subject number 14.

subject 15 (see Table IV). In his case the time of collection of all specimens was carefully controlled and noted as already described.

The urinary lead concentrations of samples taken just prior to the commencement of the thiosulphate treatment were practically the same (0·80 and 0·83 milligramme per litre) by the two methods, and again eleven days after treatment ceased (0·38 and 0·42 milligramme per litre). During the treatment there was a great difference between the concentrations found by the two methods for samples collected during the twenty-four hours subsequent to the injections, but much less difference (in the earliest stages of treatment practically none) between the concentrations in the samples collected during the twenty-four hours prior to the injections.

The urinary lead concentrations determined by the two methods for the days prior to and the days following the injections and the differences between them are shown in Table VI. Analysis of the results shown in this table indicated that there were significant differences between the values determined by the two methods for specimens of urine collected during the twenty-four hours following injections of sodium thiosulphate, but no such significant differences in the values determined for specimens collected for twenty-four hours prior to injections.

The t value obtained from a comparison of the means of the concentrations determined by the two methods for urine collected during twenty-four hours after the injections was 2·64 for the first five pairs of observations. For $n = 8$, this corresponds with a P value of 0·03, which is significant.

For the last five pairs of observations $t = 3·116$, corresponding to a P value of 0·013, which is significant.

TABLE IV.

Data for Subject 15 in Regard to Urine and Blood.

Date of Collection of Urine.[1]	Specific Gravity of Urine.	Volume of Urine. (Cubic Centimetres.)	Urinary Lead Concentration. (Milligrammes per Litre.)		Urinary Lead Concentration: Wet Oxidation. (Micrograms per Gramme of Solids.)	pH.	Blood Lead Content. (Milligrammes per 100 Grammes of Blood.)	Daily Urinary Lead Excretion. (Milligrammes.)[6]
			Taylor Method.[4]	Wet Oxidation Method.[5]				
7/6/51[2] ..	1022	810	0·80	0·83	14·33	—	—	0·67
8– 9/6/51[3] ..	1022	810	0·55	1·13	19·52	—	0·385	0·915
9–10/6/51 ..	1020	670	0·31	0·31	5·89	—	—	0·21
10–11/6/51[3] ..	1019	1110	0·36	0·54	10·80	—	0·125	0·60
11–12/6/51 ..	1018	1010	0·42	0·40	8·44	—	—	0·40
12–13/6/51[3] ..	1021	1450	0·29	0·55	9·95	—	0·16	0·797
13–14/6/51 ..	1020	1110	0·24	0·40	7·1	5·6	—	0·444
14–15/6/51[3] ..	1022	1150	0·32	0·53	9·16	5·6	0·20	0·609
15–16/6/51 ..	1020	1040	0·22	0·34	6·46	5·9	—	0·354
16–17/6/51[3] ..	1020	1300	0·23	0·60	11·4	6·0	0·24	0·78
17–18/6/51 ..	1018	1350	0·38	0·44	9·3	—	—	0·594
18–19/6/51[3] ..	1018	1450	0·24	0·31	7·1	—	0·075	0·45
19–20/6/51 ..	1013	1470	0·18	0·36	10·5	—	—	0·53
20–21/6/51[3] ..	1023	860	0·34	0·60	9·9	5·6	—	0·516
21–22/6/51 ..	1020	810	0·31	0·34	6·46	5·6	—	0·275
22–23/6/51[3] ..	1022	1070	0·35	0·41	7·08	—	0·13	0·44
23–24/6/51 ..	1022	550	0·19	0·50	8·63	5·9	—	0·275
Mean value			0·308	0·49	Total urinary lead after thiosulphate started ..			8·859
		Not collected during twenty-four hours.						
3/7/51	1021	—	0·38	0·42				

[1] Samples collected during twenty-four hours subsequent to 12 noon on first date (unless otherwise stated).
[2] Before treatment commenced (twenty-four hours' specimen).
[3] Injection of sodium thiosulphate given at 12 noon on first date.
[4] Mean value after treatment started, 0·308 milligramme per litre.
[5] Mean value after treatment started, 0·49 milligramme per litre.
[6] Total urinary lead excretion after commencement of thiosulphate injections, 8·859 milligrammes.
[7] Not collected during twenty-four hours.

If the first high pair of concentrations is omitted, the *t* for the next seven pairs of concentrations was 4·143, corresponding to a P value of 0·00125, which is significant.

For the eight pairs of concentrations the *t* value was 2·75, but the variances in the two groups of concentrations were significantly different, and hence the *t* value may have indicated this difference in the variances rather than the differences in the concentrations themselves. The *t* value from comparison of the means with the use of logarithm (*found value × 100*) as the variate was 3·315, which for *n* = 14 corresponded to a P value between 0·01 and 0·001 and about 0·006, which is significant.

TABLE V.

Data for Subject 15 in Regard to Faeces.

Date.	Date of Injection. (12 Noon.)[1]	Time of Collection of Faeces.	Weight of Faeces. (Grammes.)	Lead in Ten Grammes of Faeces.	Total Lead in Faeces. (Milligrammes.)
7/6/51		—	24·4	0·077	0·19
8/6/51		7.0 p.m.	30·3	0·098	0·30
9/6/51		4.0 a.m. / 8.15 a.m.	209·05	0·10	2·09
10/6/51		11.0 a.m.	102·25	0·07	0·71
11/6/51		5.30 a.m.	63·95	0·22	1·41
12/6/51		9.0 p.m.	38·65	0·048	0·19
13/6/51		5.30 a.m.	70·9	0·058	0·41
14/6/51		8.30 a.m.	81·7	0·11	0·866
15/6/51		—	64·95	0·066	0·43
16/6/51		7.30 a.m.	35·3	0·04	0·14
17/6/51		2.0 p.m.	61·7	0·07	0·43
18/6/51		No sample. 6.0 a.m.	38·9	0·058	0·22
19/6/51		Time not noted.	38·1	0·042	0·16
20/6/51		6.0 p.m.	27·2	0·035	0·095
21/6/51		5.0 a.m.	(only about 1 gramme sample)		
22/6/51		5.0 a.m.	54·5	0·0624	0·34
		Total lead in faeces after thiosulphate treatment started			7·791

[1] Injections commenced four days after patient's admission to hospital.

For specimens of urine collected during twenty-four hours prior to the injections the *t* value for the first five pairs of concentrations was 0·5, which for *n* = 8 corresponds to a P value between 0·7 and 0·8; this is not significant.

For the last five pairs of concentrations *t* was 2·794, corresponding to a *P* value of 0·023, which was significant.

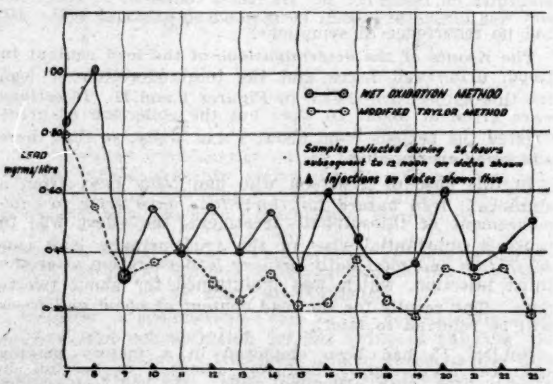

FIGURE III.
Lead in urine (Subject number 15).

For the whole eight pairs of concentrations *t* = 2·74, which for *n* = 14 corresponded to a *P* value of 0·015; this was significant. Although there is, then, a significant difference between the mean concentrations as determined by the two methods for urine collected prior to the injections, it is clear that this is not so important as it is for urine collected during the twenty-four hours after the injections.

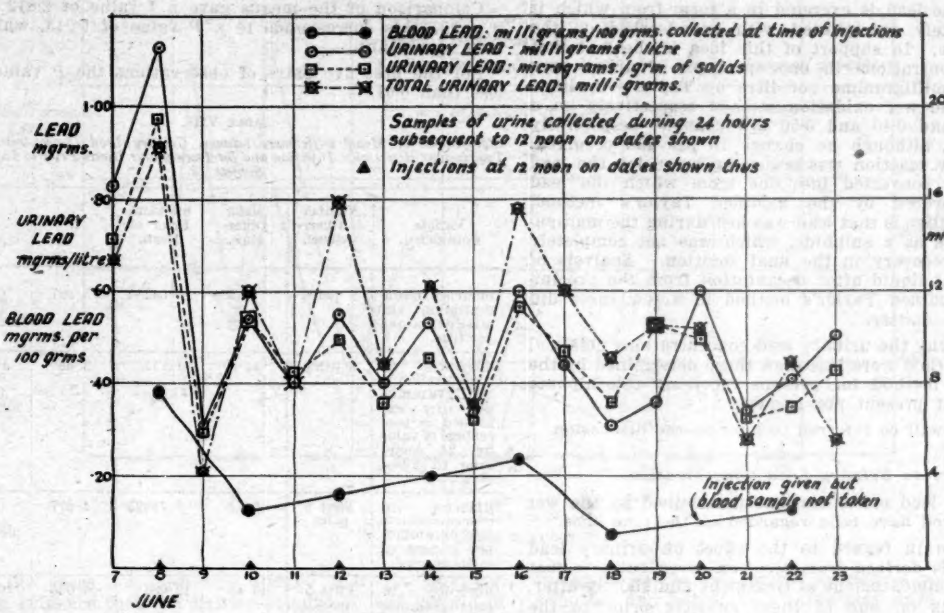
BLOOD LEAD: milligrams/100 grms. collected at time of injections
URINARY LEAD: milligrams./litre
URINARY LEAD: micrograms./grm. of solids
TOTAL URINARY LEAD: milligrms.
Samples of urine collected during 24 hours subsequent to 12 noon on dates shown
Injections at 12 noon on dates shown thus
LEAD mgrms.
URINARY LEAD mgrms/litre
BLOOD LEAD mgrms. per 100 grms
LEAD microgrm/grm. of solids
Injection given but blood sample not taken
JUNE

FIGURE IV.
Subject number 15.

In the latter case there is a significant difference between the means both for the whole eight pairs of concentrations and for the first and last five pairs of concentrations, whereas in the former case there is no significant difference for the first five pairs of concentrations.

The difference in this case is due to the differences between the last five pairs of concentrations.

Similar conclusions in regard to the earlier values are arrived at if we consider the differences between the concentrations determined by the two methods for urine collected in the twenty-four hours after, and for urine collected for the twenty-four hours before the injections. Thus the t value for the comparison of the means of the differences (fourth and seventh columns, Table VI) was 2·00, which for $n = 14$ corresponds to a P value of just over 0·05; this is not significant, although nearly so.

For the first five pairs of concentrations, however, the t value was 3·189, which for $n = 8$ corresponds to a P value of 0·012; this is significant. For the last five pairs of concentrations the t value was 0·66, corresponding to a P value of between 0·6 and 0·5, which is not significant.

Inspection of Table VI shows that in all cases the urinary lead concentrations as determined by the modified Taylor method for urine collected during twenty-four hours subsequent to the injections were considerably less than the concentrations found by the wet oxidation method. The differences were not due to any difference in the pH of the urine. It is possible that under the influence of the

TABLE VI.
Subject 15: Urinary Lead Concentrations During Course of Treatment.

Days from Start of Course of Treatment; Number of Injection.[1]	Samples Collected During Twenty-four Hours from 12 Noon on Day of Injection; Lead Concentration (Milligrammes per Litre).[2]			Samples Collected During Twenty-four Hours from 12 Noon on Days when No Injection was Given; Lead Concentration (Milligrammes per Litre).[3]		
	Modified Taylor Method.	Wet Oxidation Method.	Difference.	Modified Taylor Method.	Wet Oxidation Method.	Difference.
1. First injection	0·55	1·13	0·58	—	—	—
2.	—	—	—	0·31	0·31	0·00
3. Second injection	0·36	0·54	0·18	—	—	—
4.	—	—	—	0·42	0·40	−0·02
5. Third injection	0·29	0·58	0·26	—	—	—
6.	—	—	—	0·24	0·40	0·16
7. Fourth injection	0·32	0·53	0·21	—	—	—
8.	—	—	—	0·22	0·34	0·12
9. Fifth injection	0·23	0·60	0·37	—	—	—
10.	—	—	—	0·38	0·44	0·06
11. Sixth injection	0·24	0·31	0·07	—	—	—
12.	—	—	—	0·18	0·36	0·18
13. Seventh injection	0·34	0·60	0·26	—	—	—
14.	—	—	—	0·31	0·34	0·03
15. Eighth injection	0·35	0·41	0·07	—	—	—
16.	—	—	—	0·19	0·50	0·31
Mean	0·335	0·584	0·25	0·281	0·386	0·11

[1] The injections were given every second day at 12 noon; days are taken from 12 noon.
[2] Values for samples collected during the twenty-four hours from 12 noon on the day of injection.
[3] Values for samples collected during the twenty-four hours from 12 noon on the days when no injection was given.

thiosulphate the lead is excreted in a form from which it is not completely precipitated with or adsorbed on the calcium oxalate. *In support of this idea is the fact that* the lead concentrations in one specimen of urine were *0·19 and 0·50* milligramme per litre by Taylor's modified method and the wet oxidation method *respectively on a* certain date, and 0·40 and 0·50 milligramme respectively *ten days later, although no change in pH had occurred.* Possibly a slow reaction was occurring by which the lead compound was converted into one from which the lead could be *recovered by the modified Taylor's method.* Another possibility is that lead was lost during the manipulations, perhaps as a sulphide, which was not completely dissolved for recovery in the final solution. *Analysis of* the supernatant liquid after decantation from the precipitate *in the modified Taylor's method* in a few cases did not clarify the matter.

The reason why the urinary lead concentrations obtained by Taylor's method were less than those *determined by the* wet oxidation method for persons receiving thiosulphate *treatment is at present not known.*

This matter will be referred to later in the discussion.

Effect on Urinary Lead Concentration.

The urinary lead concentrations determined by the wet oxidation method have been *regarded as the true ones.*

The evidence in regard to the effect on urinary lead *concentration is derived from tests on seven subjects just* prior to the commencement of treatment and shortly after, and from tests on one of these subjects prior to the commencement of treatment *and daily thereafter. In this* latter case (subject 15) the collection of urine was very *carefully done, so that samples* were secured for the twenty-four hours prior to each injection and for the twenty-four hours subsequent to each injection.

The results for the seven subjects are shown in Table VII.

TABLE VII.

Subject.	Concentration (Milligrammes per Litre).		Increase. (Milligrammes per Litre.)[1]
	Prior to Injection.	Day After Injection.	
C	0·18	0·22	0·04
14	0·13	0·22	0·09
15	0·83	1·13	0·30
L	0·27	0·38	0·11
M	0·16	0·27	0·11
R	0·45	0·64	0·19
S	0·05	0·12	0·07

[1] Mean, 0·13; standard error of mean, 0·0331; *t*, 3·928; *P*, 0·01.

There was thus a significant increase in the concentrations of lead in the specimens of urine just after the commencement of the injections. This increased concentration was short-lived and was succeeded by a fairly rapid drop.

The results of statistical analysis of the findings in regard to subject 15 are recorded in Table VIII.

Effect on Urinary Lead Excretion.

The effect of the injections on the total twenty-four hour urinary lead excretion for subject 15 is shown in Table IX. The results of statistical analysis of the findings in regard to the differences between the amounts excreted in the twenty-four hours prior to, and in the twenty-four hours after, injections are shown in Table VIII. There was a significant increase in the total amount excreted in the twenty-four hours after each injection, compared with that in the twenty-four hours prior to each injection. This is especially noticeable during the first ten days—that is, for the first five pairs of observations.

Comparison of the means gave a *t* value of 2·812 when $n = 15$; this corresponds to a *P* value of 0·013, which is very significant.

For the first five pairs of observations the *P* value was less than 0·01.

TABLE VIII.

Significance of Mean Differences between Urinary Lead Concentrations for Twenty-four Hours after Injection and for Twenty-four Hours Prior to Injection; Subject 15.

Variate Considered.	Number of Observations.	Mean Difference.	Standard Error of Mean.	*t*.	*P*.
Difference in concentration in milligrammes per litre.	8 pairs.	0·164	0·04285	3·827	0·007 (significant).
Differences in concentration in milligrammes per litre expressed as percentage of value for 24 hours prior to injection.	8 pairs.	41·0	11·11	3·69	About 0·007 (significant).
Difference in concentration in microgrammes per gramme of solids in urine.	First 5 pairs.	3·71	0·79325	4·677	0·01 (significant).
Difference in microgrammes per gramme of solids expressed as percentage of value prior to injection.	First 5 pairs.	48·48	12·057	4·016	0·016 (significant).
Total lead in urine in milligrammes	8 pairs.	0·20	0·075	2·53	0·04 (significant).
Total lead urine in milligrammes	First 5 pairs.	0·33	0·05	6·54	0·005 (significant).

Fæcal Lead Excretion.

In the case of subject 14 there was a considerable increase in fæcal lead excretion above the pre-treatment values, which was maintained for about ten days. The fæcal lead excretion fell to about normal values in about twelve days.

TABLE IX.

Milligrammes of Lead in Twenty-four Hour Specimens of Urine.

For 24 Hours Prior to Injections.	For 24 Hours Subsequent to Injections.
0·67	0·92
0·21	0·60
0·40	0·80
0·41	0·61
0·35	0·78
0·59	0·45
0·58	0·52
0·28	0·44
0·28	—
Total 3·75	5·12
Mean 0·417	0·64

There is no doubt that there is a considerable increase in fæcal lead excretion subsequent to the commencement of thiosulphate treatment, but whether this is due to the treatment or to other factors is not so easy to determine. This uncertainty is due to the fact that there is frequently a great increase in the mass of fæces passed daily after the constipation has been overcome during the first few days in hospital, and it is during the first day or two in hospital that the thiosulphate treatment is commenced.

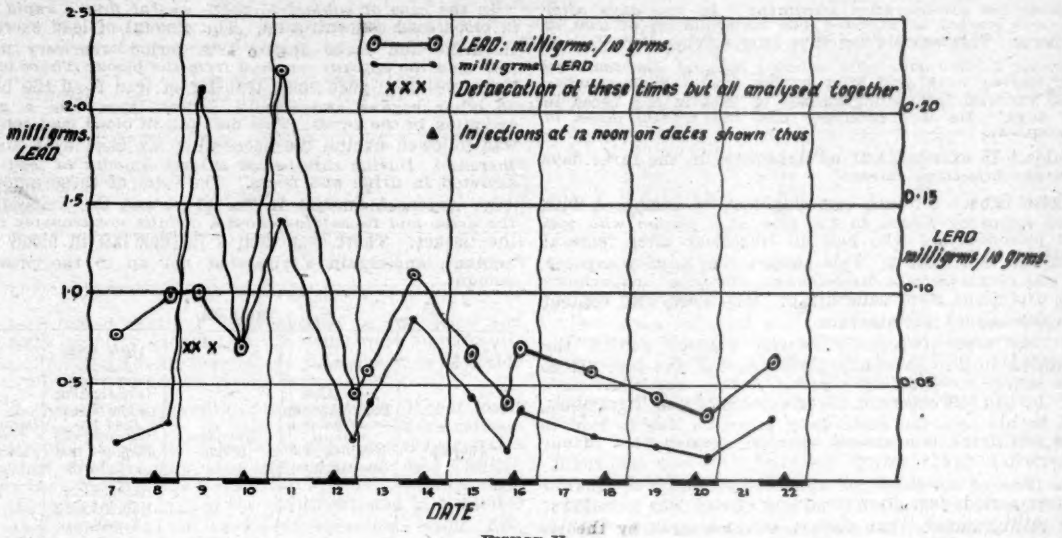

FIGURE V.
Subject number 15.

In the cases we have investigated there is, however, strong evidence that the greater amount of lead got rid of in the fæces really represents increased elimination of lead from the blood and tissues, and not merely that of lead which has been lurking in the alimentary tract.

From the point of view of fæcal lead investigation the ideal would be to have the patient undergo no treatment other than that required to secure regular and free defæcation and establish a basic level of fæcal lead excretion for a week or two prior to thiosulphate treatment, and then to commence treatment and continue the determinations. However, owing to the demand on hospital beds, the desirability of restoring the patient to health in the shortest possible time, and to the patient's natural desire for some positive treatment, such a condition of affairs can seldom be achieved.

The endeavour has been made in one or two cases, and in others the circumstances have contributed to achieving somewhat the same end.

The evidence referred to is found in the detailed results for subject 15, and in the pre-treatment history as to exposure and defæcation, and the history during treatment, of several other subjects.

Figure V shows the total fæcal lead excretion and the concentration of lead in fæces plotted against the time of excretion for subject 15. There is an obvious increase in both these quantities after the commencement of the thiosulphate treatment. The alternation of higher and lower values for both quantities is similar to that found for urinary lead concentration and total urinary lead excretion; but this alternation is not related to such definite periods before and after the time of injections. This is naturally to be expected, owing to the irregularity of the times of defæcation. On some days defæcation took place at 5 a.m. and other days at 9 p.m.

Instead, therefore, of the excretion being compared, for, say, twenty-four hours prior to and for twenty-four hours subsequent to injection, the comparison has been made between the samples of fæces which, according to the times of defæcation, might reasonably be expected to be influenced by the injection (if there is any such effect) and those which were not.

Table X shows the total fæcal lead excretion for specimens affected and not affected (or affected to much less extent) by the injection.

Determination of t for the comparison of the means gave a value corresponding to a P value of 0·035; but the variances in the two groups of figures were significantly different, and hence the t value may have determined the significance of the differences between the variances and not between the means.

The t for comparison of the means by the use of transformed values — namely, logarithm 100 (found value $0{\cdot}10 + 0{\cdot}01$)—was 2·576, which when $n = 13$ corresponds to a P value of 0·025; this is significant.

If we bear in mind that there is some uncertainty as to which specimens may have been affected by the injections and which not, it is still reasonable to conclude that the injections had a significant effect in increasing fæcal lead excretion.

TABLE X.
Total Lead in Fæces.

Specimens Affected by the Injections. (Milligrammes.)	Specimens Not Affected by the Injections. (Milligrammes.)
2·1	0·19
1·4	0·30
0·87	0·71
0·43	0·19
0·22	0·41
0·34	0·14
—	0·43
—	0·16
—	0·10

Subject 14 had been away from exposure to lead for thirty-four days prior to the start of the thiosulphate treatment. He had previously been treated for lead colic and then discharged from hospital on November 16, 1950. He returned to work on November 26, but not to the lead exposure which had produced his previous attack. He was readmitted to hospital on December 18, 1950, with colic. Thiosulphate injections were commenced on December 20, when the collection of specimens for analysis commenced. His fæcal lead excretion is shown in Figure II.

The results in this case make it extremely likely that the increased lead excretion in the fæces was due to thiosulphate treatment.

Subject H.M. had left work thirty-four days before treatment started, and had been operated on for appendicitis in hospital; he returned to work for four days, and subsequent to this had been away from exposure for sixteen days before treatment started. During this period of thirty-four days he had had an unknown number of bowel actions, including

enemata for pre-operative treatment. In two days after injections started he excreted 4·28 milligrammes of lead in the fæces. This was sixteen days after leaving work.

Subject L., five days after entering hospital, and ten days after leaving work, and after having five previous evacuations, excreted 3·66 milligrammes of lead in his fæces in two days. He was receiving half the normal doses of thiosulphate.

Subject 15 excreted 4·21 milligrammes in the three days after the injections started.

These figures for lead excretion may be compared with those found by Kehoe in the case of a person who was lead poisoned and who had no treatment after removal from the lead hazard. This subject was heavily exposed. He was very carefully investigated. His fæcal and urinary lead excretions were determined. His blood lead content was determined at intervals.

In the case of Kehoe's heavily exposed subject the excretion in any three-day period during the first fifteen days after his entering hospital, which was eight days after he had left exposure, never exceeded 3·0 milligrammes, and in his case the basic daily excretion due to lead in food and drink was almost certainly greater than in our cases.

In three of our cases the total lead excretion in equal or shorter periods just after treatment started was as follows: 3·66 milligrammes (two days); 4·21 milligrammes (three days); 4·28 milligrammes (two days).

To judge by the blood lead content and urinary lead concentration, this heavily exposed subject investigated by Kehoe was fairly comparable with our subjects in regard to the amount of lead absorbed.

If objection is raised to this comparison owing to the lack of certainty that the subjects are reasonably comparable in regard to degree of absorption of lead, useful comparison can still be made from another point of view.

In Table XI are compared the number of days after treatment or investigation commenced on which fæcal lead excretion substantially exceeded that on the first day in hospital or the day prior to treatment, for our subjects undergoing thiosulphate treatment and for Kehoe's subject having none.

There seems to be little doubt that the increased lead excretion in the fæces which follows the institution of thiosulphate therapy is actually caused by the treatment. Consideration of the changes in the concentration of lead in the fæces leads to a similar conclusion. If the thiosulphate treatment had no positive effect in increasing elimination into the alimentary tract, one would expect that the concentration of lead in the fæces would decrease as the daily mass of fæces increased. In some instances this was not so, and in all cases the increase in the mass of the fæces was very much greater proportionally than the decrease in the concentration of lead in it.

Effects on Blood Lead Content.

In the case of subject 14, who had been away from a lead hazard for some weeks prior to the first test, the effect of the thiosulphate injections was to cause a considerable increase, which reached a maximum on the sixth day. This was followed by a fairly steady fall, an approximately normal value being reached on the eighteenth day.

There was also a considerable increase in the urinary and fæcal lead excretion above the pre-treatment values, which was maintained for about ten days.

The fæcal lead excretion fell to approximately the normal value in about twelve days; but the urinary lead excretion continued at higher than the pre-treatment level for another six days, at which time treatment and tests ceased.

There was no recurrence of symptoms during the period of higher blood lead content, or at any other time during the treatment.

The number of fæcal lead determinations was not sufficient to allow a balance to be struck between loss of lead from the blood and the total lead excretion in urine and fæces.

In the case of subject 15 there was at first a rapid fall in blood lead concentration. The amount of lead excreted in urine and fæces during this period was very much less than the amount removed from the blood. There must, therefore, have been some transfer of lead from the blood to other organs and tissues, unless there was a great excretion in the sweat. This first fall in blood lead content was followed during the succeeding six days by a steady increase. During this period a large amount of lead was excreted in urine and fæces. The total of these amounts (the increased amount in the blood and the amounts in the urine and fæces) indicated a definite net transfer from the tissues. There was then a further fall in blood lead content, and again a rise, but not up to the previous amount.

TABLE XI.

Subject.	Days After Final Exposure when Subject Entered Hospital and Investigation Began.	Treatment Given.	Days After Start of Treatment or Investigation when Fæcal Lead Excretion Greatly Exceeded that on First Day in Hospital or Day Prior to Treatment.	Duration of Treatment. (Days.)
Kehoe's heavily exposed patient.	8	None.	Never in first 12. Twice in first 30.	—
Present investigation:				
Subject 14	18 (injections started 2 days later)	Thiosulphate.	Four out of first 6.	12
Subject 15	1 (injections started 4 days later)	Thiosulphate.	Seven out of first nine.	15
Subject H.M.	15	Thiosulphate.	Two out of first five.	9 (specimens secured for only 3 days after treatment started)
Subject L.	3 (treatment started 5 days later)	Thiosulphate (half doses).	One out of first four (fæces of 2 days not supplied)	5 (in hospital)

An overall balance during the period of treatment indicated that the amount of lead lost in urine and fæces was just about equal to the amount lost from the blood.

There is thus no storage of lead brought about by the thiosulphate treatment in tissues other than the blood. Since any excretion via the sweat has not been considered (and unpublished work so far done indicates that the amount so excreted may be considerable), there is no doubt that in this case the effect of the thiosulphate was to increase the elimination of lead.

Table XII summarizes the various effects just mentioned. It has been assumed that the weight of the blood was 8% of the body weight, which was 10 stone 11 pounds, and that the daily excretion of lead in the fæces due to ingestion in food and/or water was 0·075 milligramme.

If we consider the blood lead contents after a few days of treatment, it is found in all cases that there is a significant fall. At the end of nine days of treatment the falls in four cases were 0·16, 0·10, 0·12 and 0·22 milligramme; $t = 5{\cdot}67$ when $n = 3$; $P = 0{\cdot}008$.

DISCUSSION.

It has been shown by comparing the urinary lead concentration just prior to the commencement of treatment and on the next day for different subjects that an increase occurs. It has also been shown in the case (subject 15) in which the time of collection of specimens was very

TABLE XII.

Loss and Gain of Lead from Blood and Body (Milligrammes).

Period.	Loss of Lead from Blood.	Gain of Lead in Blood.	Loss of Lead from Body.		Total Loss.	Gain by Tissues.	Loss by Tissues.
			In Urine.	In Fæces.			
8–10/6/51	14·30	—	1·125	2·95	4·075	10·225	—
10–16/6/51	—	6·325	3·207	3·055	6·262	—	12·587
16–18/6/51	9·075	—	1·374	0·5375	1·9115	7·1635	—
18–22/6/51	—	3·025	1·771	0·370	2·141	—	5·166
Total	23·375	9·350	7·477	6·9225	14·3895	17·3885	17·753

carefully noted that the concentration in the urine and the total quantity excreted in the urine were significantly greater for the twenty-four hours subsequent to injection than for the twenty-four hours prior to it.

This increased urinary lead concentration is not maintained for as long as is the case with sodium citrate treatment. In the latter instance the increased excretion may last for a considerable time. In one case (Shiels, 1950) the excretion was maintained at a high level throughout the first thirty days of the treatment, and in a number of other instances the increased excretion lasted five, seven, ten and nineteen days.

The difference in the dosages may be the reason for this. The daily average dose of thiosulphate was one gramme (two grammes every second day), whereas the dose of the citrate was 12 to 15 grammes daily. Thus it is hardly to be expected that the thiosulphate would have such a great or so prolonged an effect as the citrate.

However, clinical experience indicates that the thiosulphate treatment is as effective as the citrate treatment, and it may be that the thiosulphate has an important function in supplying sulphur. In this connexion it is pointed out that sulphur in various other forms has been useful in the treatment of lead poisoning. Sulphuretted hydrogen, sodium tetrathionate, sulphur baths, even BAL (in certain stages of the disease) have all been used successfully for this purpose.

In regard to subject 15, there was a change in the relationship between the excretion or concentration of lead in the urine on the days before and after the injections in the later stages of the treatment.

There is, of course, no *a priori* reason for assuming that the effects of the injection have passed away before the second twenty-four hours after the injection; the results in the early stages of the treatment suggest that they have, but those in the later stages that they have not.

Similar conclusions are suggested by the consideration of the differences between the results of analysis by the two different methods for the day before and the day after the injections, which have already been mentioned.

There appear to be two factors operating.

In the early stages of the treatment, during the second twenty-four hours after injection—that is, during the twenty-four hours prior to the next injection—the results obtained by the two methods agree very closely, which indicates that the thiosulphate has been all used up or excreted during the first twenty-four hours after the injection.

During the later stages of the treatment the results obtained by the two methods for the twenty-four hours prior to injection, instead of agreeing as at first, tend to differ considerably, which indicates that there is probably a gradual storage of thiosulphate in the body, or a less rapid elimination of it. On the other hand, in these latter stages the differences between the results obtained by the two methods for the urine collected during the twenty-four hours subsequent to injection tend to grow less.

These statements apply whether the differences are expressed in actual concentrations or as a percentage of the wet oxidation value.

For urine collected during twenty-four hours after the injections, the average differences in the urinary lead concentrations determined by the two methods expressed as percentages of the wet oxidation values changed from 46·6% for the first ten days of treatment to 27% for the last six days.

For urine collected during twenty-four hours prior to the injections, the corresponding figures changed from 18% for the first ten days to 40% for the last six days.

It may be that in the earlier stages of treatment the lead, or a considerable proportion of it, is in a form with which the thiosulphate readily reacts, whereas in the later stages considerable time is required for the thiosulphate to combine with the more firmly bound lead.

At the present stage of our knowledge it is considered inadvisable, in the case of patients undergoing treatment, to rely on methods for the analysis of urine for lead which depend on precipitation from the original urine. It should not be assumed that a method which is reliable for the urine of persons not undergoing treatment will also be satisfactory while they are having treatment.

The preferred method of analysis in such cases is one in which there is complete oxidation of evaporated urine.

SUMMARY.

1. The intravenous injection of sodium thiosulphate results in a difference in the urinary lead concentrations determined by a modified Taylor's method and those determined by wet oxidation followed by dithizone mixed colour determination, the latter values being much the higher. The reason for this is not at present known.

2. There is a significant increase in the true urinary lead concentration and the total urinary lead excretion on the first day of treatment.

3. There is a rapid fall in this concentration and excretion in the next few days.

4. In the case in which the times of collection of samples relative to the times of injection were carefully noted there were a greatly increased concentration of lead and a greatly increased total excretion in the urine for the twenty-four hours subsequent to each injection, compared with those for the twenty-four hours prior to each injection.

5. It is almost certain that the treatment increases the fæcal lead excretion.

6. The treatment causes a significant fall in the blood lead concentration with, in some cases, a temporary rise.

7. There is no evidence that the treatment causes any storage of the blood lead content in organs or tissues.

ACKNOWLEDGEMENTS.

Acknowledgements are gratefully made to Dr. R. Smibert, superintendent of the Alfred Hospital, and to Dr. J. H. Lindell, superintendent of the Royal Melbourne Hospital; to the late Dr. D. Thomas, formerly honorary physician to the Royal Melbourne Hospital, to Dr. K. Fairley and Dr. W. McI. Rose, honorary physicians to the Royal Melbourne Hospital, and to Dr. Eric Clarke, honorary physician to the Alfred Hospital, for access to patients and clinical records; to the nursing staffs of the Royal Melbourne

Hospital and the Alfred Hospital for the collection of specimens; to Mr. R. T. Leslie, of the Department of Mathematical Statistics of the University of Melbourne, for the statistical analysis of the results recorded in Table III and the conclusions based thereon; and to Miss J. Harwood, of the Department of Health, for the preparation of the graphs.

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A MALARIA SURVEY OF THE PEOPLE LIVING ON THE MINJ RIVER IN THE WESTERN HIGHLANDS OF NEW GUINEA.

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A LONG time ago two women came to Minj, where the government station is now, from Wilumbuga on the road to Mount Hagen. They burned the kunai grass at Minj and gathered the wild food exposed by the fires. These two women built a house for themselves and started a garden. They soon found that the food was disappearing from their fireplace while they were away in their garden. One day they hid in the house and saw two dwarfs, one stealing the food from the fireplace and handing some of it to the other who stood watch at the door. The women rushed out and caught the dwarf at the fireplace and tied him to a pole in the house. The second dwarf ran off to some distance and shouted out to the captive to turn himself into a pig or a snake; but this did not happen, and the second dwarf ran away. The captured dwarf was untied and stayed in the house, where he was made to cook and look after the house.

One day news came that there was to be a dancing feast at Nondugal. The two women dressed themselves up and went off to it. While they were away the dwarf cooked all their food and gave some of it to his friends. The rest he took with him to his own place, where he changed himself into a true man and also went to the festival with the food under his arm. The stranger was seen by the two women, who thought he must have come from another place; but they saw also the food under his arm. One of the women ran home and found the food all gone; but when she came back with this news the stranger ran off, and, changing himself back to a dwarf, returned to the women's house, where he was again tied to a pole.

The two women married the dwarf, and they both had babies. The first babies were the trees which the Minj people now use to make their broad belts. The second babies were an aromatic shrub. The third babies were normal boys called Singa and Angin. The navel-strings of these two baby boys were planted in the earth, and one came up as a banana tree and the other as a native bean. Before this there were no bananas or beans. When the banana tree bore fruit the women did not think it would be good to eat, as they knew whence it came. At night they saw a flying fox eating the banana fruit, and they did the same and found it was good. They planted out the suckers of the banana tree. The beans were also found to be good to eat.

When the two boys grew up, Singa married a girl from Nondugl and had two sons, Korup the elder, and Page the younger. Angin also married and his children went over Banz way. Korup, the elder son of Singa, married a girl from Konoka, and his children and their children are the people of the Minj Valley.[1]

General Information.

In May, 1954, a malaria survey of the reconnaissance type was made of the people living along the Minj river in the western highlands of New Guinea. The Minj river has its source in the Kubor Range, which forms the southern wall of the Waghi Valley,[2] and runs to the north-west to join the Waghi river. Malaria is present to a high degree in the Waghi Valley (Christian, 1949-1951; Giblin, 1954), and the object of this survey was to determine whether transmission occurred on the mountainous walls of this valley.

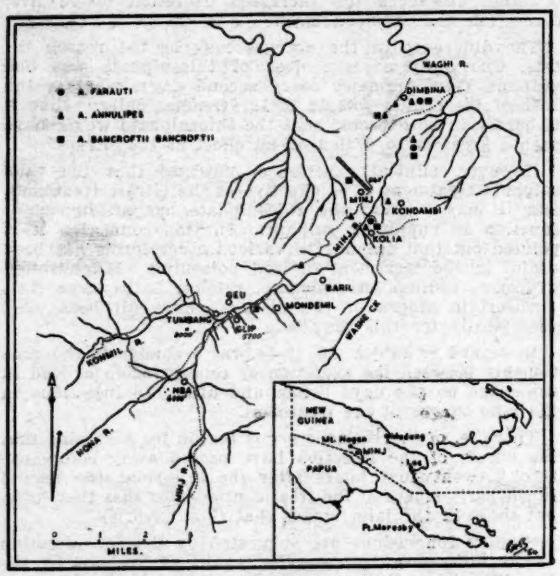

FIGURE I.

The Minj river, showing localities and the sites at which various species of anophelines were found. The inset shows the general location of Minj.

Ecology.

The Waghi Valley is about 60 miles long and from five to 15 miles wide. It is bounded to the north by the Bismarck Range and to the south by the Kubor Range, which rises to heights of 13,000 feet at Mount Sigal Mugal and 12,500 feet at Mount Kubor. The floor of the Waghi Valley is generally flat. The Minj river, with its origin in the southern wall of the Waghi Valley, flows for three-quarters of its length through the narrow, steep-walled Minj Valley. In the last quarter of its course the Minj

[1] This story was related by Nop Nop in consultation with some old men of the Minj Valley.

[2] Various methods of spelling "Waghi" have been published. The present method is taken from the map of the Minj Administrative Area prepared from compass traverses by G. T. Linsley, upon which the accompanying map (Figure I) is based. Giblin (1954) used the spelling "Whagi", and Gilliard (1953) "Wahgi". Phonetically there appears to be no necessity for the "h", and Vial (1941) omits it.

river has cut a broad U-shaped valley in the floor of the Waghi Valley. Here, the river pursues a more meandering course than that part in the Minj Valley itself. In the Waghi Valley the left bank of the Minj river falls away, but the right bank continues as a low spur to the junction with the Waghi river. Numerous tributaries flow into the Minj river in its course through the mountains, and Wasne creek enters as it emerges into the Waghi Valley. Minj, the Government station for the area, is on the left bank of the lower part of the Minj river and is 5140 feet above sea level.

The mountainous walls of the Minj Valley are well wooded, except where large areas have been cleared for gardens by the inhabitants. In the gardens are grown sweet potato (the staple food), bananas, taro, sugar cane, paw paw, peanuts, potatoes, sweet corn, beans and greens. Several of these have been recently introduced. The gardens are divided by drains into squares of about six feet.

FIGURE II.
The Minj river in the lower part of its course in the Waghi Valley.

Observations have been made at Minj on the climate over the year 1953-1954. There appear to be definite wet and dry seasons. Table I gives rainfall, temperature and relative humidity figures for the year May 1, 1953, to April 30, 1954. It will be seen that the wet season extends from November to April or May. The nights are cold and humid and the days warm and dry. These observations at Minj station probably apply to the Minj Valley.

The Waghi Valley was first entered by Europeans in 1932, when it was patrolled by Taylor and Black—officers of the administration—and the Leahy brothers came seeking gold. The airstrip was built at Minj in 1943, and after this there was a Government post manned intermittently until 1952. Since 1952 Minj has been a stable subdistrict centre. The Waghi Valley people are somewhat smaller than the average European in height, but the

FIGURE III.
The Minj river in the upper part of its course showing the rapid rate of flow.

adults are muscular. The males are picturesque in appearance, wearing a plumed headdress, shells through the nose, painted faces, and a broad belt with a curtain of open weave netting hanging down in front and a bustle of leaves behind. The unmarried girl is also gaily dressed, but the married woman's dress is simple. Gilliard (1953) has published numerous coloured photographs of the Waghi Valley people seen at Kup.

Living in the Minj Valley are 3300 people, and there are a further 300 on the lower Minj, which lies in the Waghi

TABLE I.
Rainfall, Temperature and Relative Humidity Observations at Minj for the Period May 1, 1953, to April 30, 1954.

Month.	Rainfall.		Average Temperatures. (Degrees Fahrenheit.)			Relative Humidity. (Percentage.)		
	Points.	Rain Days.	Minimum.	Mean.	Maximum.	8 a.m.	12 noon.	4 p.m.
May	528	12	54	62	80	84	47	47
June	472	10	58	70	82	87	46	48
July	594	18	56	68	80	88	51	54
August	362	16	56	66	80	90	47	50
September	1081	22	55	63	81	91	44	49
October	471	21	56	69	82	88	44	48
November	839	24	56	68	80	90	53	60
December	853	18	56	68	80	90	48	52
January	1259	30	58	69	80	93	50	51
February	1115	21	56	68	80	91	52	60
March	1096	27	58	69	80	92	53	62
April	1164	23	58	70	82	92	53	57
Total ..	9934	242	—	—	—	—	—	—

Valley. The people are farmers and live in very small scattered hamlets or isolated houses where their gardens are growing at the particular time. The gardens last for up to one and a half to two years, and then the site is changed. The houses are built with the ground as the floor and have a low thatched grass roof. The walls are also made from grass piled in between upright wooden supports. The result is a hot-box with a smoking fire inside. The larger houses are divided into three compartments. The first is near the door, and in this is the fireplace where cooking is done. The central compartment is the sleeping area for the pigs. At the rear of the house the women and children sleep. The men sleep alone in round, single-roomed houses. On occasions families take their pigs from the Minj Valley to the lower Minj or Waghi Valley, and there graze them for periods which

FIGURE IV.
Mountainous walls of the Minj Valley.

may be as long as two years. Apart from pigs, few domestic animals are kept. Fowls and dogs are rare. Occasionally a bird or opossum is kept for a while before disposal.

The following are common diseases in the Waghi Valley: infections of the respiratory tract; yaws, which does not appear to be so florid as when seen on the coast; endemic goitre; leprosy; hookworm; diarrhœal diseases; and scabies. Injuries sustained in family brawling are common. No reliable figures are yet available for birth, death and infantile mortality rates.

At Minj Government station there is a recently established native hospital. Minj is also the site of the Malaria Control School of Papua-New Guinea, and here, too, is being carried on the Waghi Valley Pilot Drainage Scheme, which was started at Banz on the north side of the Waghi river in 1951. There are two medical aid posts situated on the Minj river, but these do not as yet appear to have been popularly accepted by the Minj river people.

Malaria in the Waghi Valley.

In the Waghi Valley four species of anopheles occur: *A. farauti*, *A. punctulatus*, *A. annulipes* and *A. bancroftii bancroftii*. Christian has found both *A. farauti* and *A. punctulatus* infected in the Waghi Valley. Malaria in the Waghi Valley appears to be seasonal, occurring mainly between February and May, as judged by patients presenting themselves for treatment at the Minj hospital. Three species of malaria parasite have been found, *Plasmodium falciparum*, *P. vivax* and *P. malariæ*.

Malaria in the Minj River People.

It is convenient to divide the results of the survey of the people living along the Minj river into two parts, upper Minj and lower Minj, the upper Minj being that part of the river flowing through the mountains, and the lower Minj being in the Waghi Valley.

The people of the upper Minj were examined at three locations: Baril, Glip and Uinba. At these locations the people of Geu, Kumanga, Tumbanga and Mondemil were also seen. Palpation of the spleen and examination of the blood for malaria parasites were carried out on a total of 428 people seen in the upper Minj. The results are presented in Table II.

Of the 41 people with palpable spleens in this group, 39 had lived in the Waghi Valley; some were Waghi Valley people visiting the Minj people, some women were born

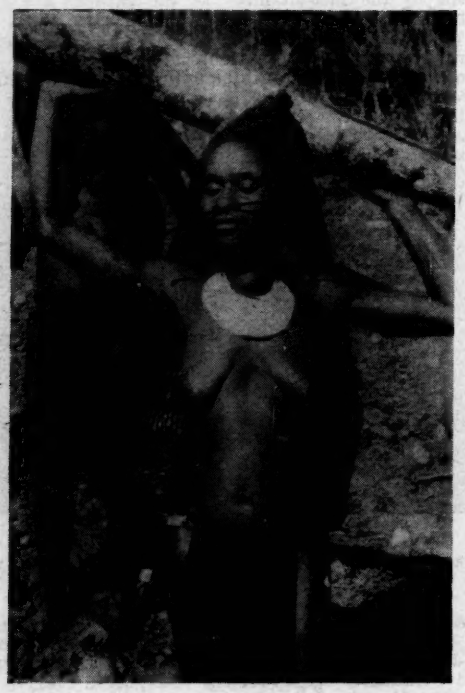

FIGURE V.
Woman seen on lower Minj with endemic goitre.

there and had married Minj men, others had lived in gardens and/or kept pigs in the Waghi Valley, whilst others had been to school there. Two children were examined, one aged ten years at Baril, the other aged six years at Uinba; both had size I spleens, and, it was stated, had never been away from their homes. No malaria parasites were seen in the blood films made from these two children.

It will be noted that the numbers in the age groups indicate that the younger children did not present themselves for examination as readily as the older people. No new-born babies were examined, not were any pregnant women. No attempt was made to overcome this reticence, as it was considered that sufficient information was obtained from those who had been examined.

In the upper Minj area a wide search was made for anopheline larvæ, and *A. annulipes* only was found in two locations: at Alua where the track crossed a small seepage, and near the aid post near Kumanga in a blocked drain containing clear water with some emergent grass and general light shade. In many places along the track pools were formed in depressions and were broken up by footmarks. In addition, seepages were seen at the edge of the road; some of these had been blocked off to form pools. Several of these seepages were covered by a film associated with the growth of iron bacteria.

TABLE II.

Results of Spleen and Parasite Examination of Upper Minj People.[1]

Age Group. (Years.)	Number of Persons Examined.	Palpable Spleens.						Malaria Parasites.		
		I.	II.	III.	IV.	V.	Total.	F.	V.	M.
0 to 1	1	—	—	—	—	—	0	—	—	—
2 to 10	68	3	1	—	—	—	4 (6%)	1	2	1
11 to 20	145	7	7	3	1	—	18 (12%)	5[2]	5[2]	1
21 to 30	95	4	4	—	—	—	8 (8%)	—	—	—
31 and over	119	6	4	—	—	—	10 (8%)	2	—	1

[1] Spleen sizes are recorded as recommended by Hackett (1944). Spleens were palpated with the native recumbent. "F", *Plasmodium falciparum*; "V", *P. vivax*; "M", *P. malariæ*.

[2] Includes one mixed infection.

Search of the houses in the Uinba and Baril area in the daytime did not reveal any anophelines. A night catch (one hour, three catchers using themselves as bait) was made at Kumanga and no mosquitoes were seen. A night catch (one hour, one catcher using himself as bait) at the aid post near Kumanga resulted in the capture of five culicines and no anophelines.

In the lower Minj area 134 inhabitants were examined at Dimbina and at Kondambi, where people came from along the ridge forming the right bank of the Minj between Kondambi and the Waghi river. The results of spleen and parasite examinations are presented as Table III.

Figure VI.
New house at Dimbina on the lower Minj.

Search by day of some of the houses occupied by these lower Minj people resulted in the catching of 15 female *A. farauti* (14 engorged), 12 female *A. bancroftii bancroftii* (all engorged), and one female *A. annulipes* (engorged). Night catches were made at Dimbina near the left bank of the lower Minj close to its junction with the Waghi, and at Kolia, which is on the right bank of the Minj about half a mile down from the junction of the Wasne creek with the Minj river. At Dimbina four catchers with four bait between 8 p.m. and 9.30 p.m. caught 61 *A. farauti*, nine *A. bancroftii bancroftii* and one *A. annulipes*. At Kolia four catchers with three to six bait between 8.30 p.m. and 10 p.m. captured 22 *A. farauti*, seven *A. bancroftii bancroftii* and three *A. annulipes*. The night catch at Dimbina provided material for dissections. A total of 43 *A. farauti*, and eight *A. bancroftii bancroftii* were dissected, but none were found to be infected.

A. farauti larvæ were found in a seepage area in the kunai grass at Dimbina. Numerous breeding sites of *A. annulipes* were found in blocked drains along the road and in a garden, in depressions on the road, and in water collections in gardens. The breeding sites of *A. bancroftii bancroftii* were not found. It will be recalled that a drainage project is being carried out near the Government station, and in addition oiling squads of natives using DDT in power kerosene are active on both banks of the lower Minj river.

FIGURE VII.
Foot road in the Minj Valley, showing potential anopheline breeding sites.

Discussion.

Malaria amongst the people on the lower Minj river is probably hyperendemic, although insufficient children in the age group two to ten years were examined to place the degree of malaria in this category rather than in the upper range of mesoendemic malaria. Also, the survey was made at the end of the transmission period when spleen rates would tend to be high.

In the upper Minj it is extremely doubtful whether any transmission of malaria occurs at all. The only anopheline found was *A. annulipes*, in considerable contrast with the numbers of *A. farauti* found where the lower Minj flowed through the Waghi Valley. Gametocyte carriers were

TABLE III.

Results of Spleen and Parasite Examination in the Lower Minj People.

Age Group. (Years.)	Number of Persons Examined.	Palpable Spleens.						Malaria Parasites.		
		I.	II.	III.	IV.	V.	Total.	F.	V.	M.
0 to 1	2	1	—	—	1	—	2	—	1	—
2 to 10	26	6	6	3	2	—	17 (65%)	5	1	—
11 to 20	58	13	23	10	1	—	47 (81%)	8	3	4
21 to 30	23	2	7	7	—	1	17 (74%)	1	—	—
31 and over	25	3	3	9	1	—	16 (64%)	1	—	—

shown to occur either among people coming to live in the Minj Valley from the Waghi or among Minj people returning from a period of residence in the Waghi Valley. Thus there is opportunity for transmission if a suitable vector is present. Two children were found with slight splenic enlargement for whom a period of residence in the Waghi Valley could not be established. It is doubtful whether the possibility of casual residence, such as an overnight visit,

FIGURE VIII.

Another part of the foot road in the Minj Valley showing potential anopheline breeding sites.

can be excluded. The other possibility is transmission by *A. annulipes*. From a practical point of view the great contrast between malaria in the upper Minj and lower Minj people is remarkable. Control measures need not be applied to people living in the Minj Valley.

This distribution of endemic malaria corresponds exactly with the configuration of the land in which the two groups of people live. In the upper Minj they have their houses on the steep sides of the Minj Valley, which rise up to more than 2000 feet above the river bed at Uinba. The river itself drops almost 1000 feet in a distance of about five miles between Uinba and its entrance to the floor of the Waghi Valley. In the lower Minj the ground is much flatter, seepages occur in the kunai grass, and roads have been built with inefficient drains, so that breeding sites are provided.

Amongst the Waghi Valley people as a whole (and in other parts of the highlands) there is a traditional belief that bad spirits, which are active at night, inhabit the valley especially near the rivers. These spirits in the distant past drove the people from the valley floor to the mountainous slopes of the valley walls. One of the manifestations of the influence of these spirits was fever with ague. The spheres of influence of these spirits were well defined areas in the valley floor, and were pointed out to Taylor (1954) when he first came to the Waghi Valley. Taylor's party suffered from attacks of fever during their patrol in the valley. The Waghi people also believed that some people could come to terms with the bad spirits and could live near these spheres of bad influence without undue harm. In the case of those not so favoured, a distance of three miles was necessary for protection.

"*Koronum*" is the local name for fever with ague, which the inhabitants recognize now as "malaria". Their traditional treatment was carried out by a medicine man ("*Kunderonim*"), and consisted of beating the patient all over with nettles and the playing and then burning of a bamboo flute. When the feverish attack was over, a pig was killed and its liver cooked and the medicine man made passes over it. The liver was then fed to the patient. If the patient recovered, his attending physician was paid for his services.

During this survey, when it appeared probable that no malaria transmission occurred in the upper Minj people, they were questioned on the incidence of malaria in the area. They were quite emphatic that no malaria attacks had their origin in the Minj Valley. The disease occurred only in those who went to live in the Waghi Valley or who had recently returned from that area. There was much less certainty about the mortality rate due to the malaria acquired in the Waghi Valley—an estimate was given of two in ten. It was stated that people of all ages had died as a result of this illness.

With regard to the question why malaria—that is, the vectors *A. farauti* and *A. punctulatus*—has not penetrated the Minj Valley when it is present at its very mouth, the only tenable explanation is that the drainage on the slopes of the valley wall and a narrow valley floor with a considerable slope downwards in the direction in which the river flows, have provided adequate drainage in the past and thus denied the vector suitable breeding places. In addition, some seepages were seen with scum on the surface, associated with growth of iron bacteria, which are unsuitable breeding sites for mosquitoes. Altitude, in itself, has not limited the extension of *A. farauti* into the valley, as this species has been collected by Christian at Wabag at a height of just under 7000 feet. The Minj Valley appears to be vulnerable to penetration by vector anophelines via the foot road. The accompanying photographs (Figures II to VIII) show potential breeding sites for vector species along this road in the Minj Valley.

This survey has shown that a group of 3000 people are living in an area where malaria is not endemic, and which is only a few miles removed from an area where malaria is probably hyperendemic. This sudden change is associated with the configuration of the land. Further surveys may allow of a general statement about transmission on the slopes of the highland mountains. In this case it appears that the sloping nature of the country has denied

the vectors breeding sites in the area; but there is a possibility that man-made breeding places may enable vector species to become established.

Acknowledgements.

The writer is grateful to the Director of Public Health, Papua and New Guinea, for the opportunity of visiting this area, and to Mr. S. H. Christian for hospitality, information and the use of the facilities of his laboratory and staff. Officers of the Administration provided information and hospitality, for which gratitude is expressed. The blood films were stained and examined by Mr. A. L. Gray, of the School of Public Health and Tropical Medicine, Sydney. The paper is published with the permission of the Director-General of Health, Commonwealth Department of Health, Canberra.

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INDUCTION OF LABOUR.

By E. KENT-HUGHES,

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I HAVE used a simple method of induction of labour for many years, and it is so universally successful that I think that it is worth recording. The only contraindication to the use of this method is the presence of a tightly closed, stenosed cervix.

There are many occasions when induction of labour is required, and I think that post-mature and large babies are the cause of many injuries to the mothers, and the children themselves are often injured at birth. Therefore labour is induced at term, or, if the baby is large, labour may be brought on fourteen days or so before the calculated date.

Labour is also induced if the head is not fitting well into the pelvis, if toxæmia is present, or if Rh-negative mothers have antibodies in the blood. Also women in the country districts will come into hospital if they know that their stay there will not be unduly prolonged. Thus are avoided the dangers encountered by women in labour in a rushed trip over country roads. Another reason for induction of labour is the occurrence of weak, irregular and intermittent uterine contractions which tend to come and go for days. These contractions are very tiring and make the mother nervous and unfit to cooperate when her time of trial really arrives.

The average weight of the babies in a series of 85 cases of induction of labour was seven pounds three ounces. The low application of forceps was used twice, and an episiotomy was often performed on primiparous patients. Two babies were stillborn; one case was a difficult breech presentation, and one child was strangled with a cord knotted tightly round the neck.

The ordinery routine procedure is as follows. One ounce of *Oleum Ricini* is given at 6 a.m. Quinine sulphate, 10 grains, is administered at 7 a.m. and the membranes are ruptured at 8.30 a.m. The same dose of quinine is repeated at 9 a.m. and again at 11 a.m. if labour pains have not commenced; this is needed in only about 15% of cases.

When the membranes are ruptured, the cervix is stretched as far as possible and the membranes are stripped if the cervix will admit the finger. Then an ordinary steel knitting needle is used to puncture the membranes. An anæsthetic is rarely needed.

The average delay before delivery from the time the membranes were ruptured is ten hours. The shortest time was three-quarters of an hour and the longest fifty hours. There were six protracted cases in which the period varied from twenty-eight to fifty hours, with an average of thirty-eight hours. If these were classified separately, the average period in the others would be eight hours.

There were no complications such as infections, and all the babies, excepting the two mentioned, did well.

I do not use "Pituitrin", as I had that drug given to me once. The baby took a week to arrive after three series of treatments, and I shall never forget the pain of the violent, ineffectual uterine contractions. I am convinced that the method is dangerous.

The method of induction that I have outlined brings comfort to the mothers and safety to the babies, and saves the doctor much night work.

THE USE OF THE RESECTOSCOPE IN CONDITIONS OTHER THAN PROSTATISM.[1]

By LEONARD MURPHY,

Melbourne.

THIS paper consists of a brief account of those conditions other than prostatism in which the use of the resectoscope provides the best, and in some the only, method of treatment.

The prostatic electrotome, to call this instrument by its catalogue title, resulted from hundreds of years of effort directed towards removing the obstructing prostate by the transurethral route. Ambroise Paré's sound with its terminal knife was one of the earliest recorded attempts. The invention of the cystoscope and development of the coagulating current caused a renewed but unsuccessful attack on the prostate; but from this failure Beer originated the fulguration treatment of bladder tumours. In 1925 the first reports of the use of the cutting current on the prostate were published and the basis of the modern instrument was established. Attention for the next fifteen years or so was focused on excision of the prostate, and it is only in the last decade that the use of the resectoscope in the treatment of bladder tumours, the neurogenic bladder and other lesions has been developed and is being evaluated. I have been unable to find any landmarks in the enlargement of the scope of the instrument. It appears that as surgeons have developed a satisfactory technique in excising the prostate, the use of the instrument in other accessible conditions has followed naturally. The spinal injuries during the recent war have established the role of the resectoscope in the treatment of the neurogenic bladder.

Bladder Neck Obstruction in the Female.

Bladder neck obstruction is a disease of women past middle age; two of our best results have been obtained in women aged over eighty years. The condition is not uncommon; but because the syndrome is not so definite as in the male, nor is a pathological basis so easily ascribed to it, it has been rather neglected. This condition was fully discussed by Davidson at a previous meeting. The patient complains of frequency of micturition and of scalding and some dysuria of varying degree. Retention of urine may occur, but is not common, and rarely the full bladder of retention with overflow is found on examination. The intravenous pyelogram usually reveals no abnormality. Cystoscopic examination shows trabeculation to be present, though not necessarily pronounced, and there may be nothing obviously obstructive in the bladder neck. Residual urine is present in varying amounts, and unless one remem-

[1] *Read at a meeting of the Urological Society of Australasia on April 20 to 23, 1953, at Surfers' Paradise, Queensland.*

bers the possibility of the condition and looks for residual urine, one is apt to overlook the diagnosis and label the condition as recurrent urinary infection. Of the series of 76 cases of Emmett and his co-workers, although in 70 residual urine was present, dysuria was a symptom in rather less than 50%, frequency of micturition being the most common complaint. Powell and Powell have achieved good results with resection of the bladder neck in women with mainly irritative bladder symptoms and no residual urine; but unless more definite indications than these were required, one might operate on a large proportion of the female population.

The ætiology and pathological basis of the condition are still not understood. Emmett found no evidence of glandular tissue in his cases sufficient to cause obstruction, and the "female prostate" theory now appears to be untenable. Examination of the sections shows smooth muscle, at times hypertrophied; rarely is fibrous tissue a feature; and inflammatory cell infiltrations, though present, do not appear to be of causative importance.

The treatment of these patients is by transurethral resection. The proximal third of the urethra is safe for resection, and tissue is taken around the circumference of the bladder neck. Resection of the anterior aspect of the bladder neck is just as important for a good result as the posterior and lateral sectors usually dealt with in the male. The depth of resection varies from case to case, and judgement comes with experience. A second resection presents no difficulty if a good result is not obtained at once, and is frequently necessary. Such repeated resections are more easily performed and more sure to be successful than attempts at curing incontinence of urine resulting from cutting too deeply, in an attempt at an immediate good result at the first operation.

Bladder Neck Obstruction in Children.

Instruments of small calibre have been developed which enable resection of the bladder neck to be performed in very young children—the infant resectoscope is of 16 French calibre and the cold punch 18 French. In the younger males, perineal urethrotomy is advisable to avoid stricture formation.

Bladder neck obstruction in children may be divided into the following three main groups: (i) The neurogenic bladder group. The treatment of this follows the same pattern as that described later for the adult. The majority of cases at our clinic have been of the autonomous type due to *spina bifida*, and good results have followed resection. (ii) The presence of valves in the posterior part of the urethra. This is the best known entity, and though it has been described by some, such as Dodson, as the most frequent cause of lower urinary tract obstruction in children, it is not commonly found in practice. Up to 1934 there were only 130 reported cases. Emmett and Helmholtz found no examples in 31 cases of bladder neck obstruction in children. This condition can be treated by transvesical or retropubic operation, but equally good results are obtained by transurethral incision, fulguration or resection. (iii) Other cause group. This heading includes hyperplasia of the child's prostate, median bars, and contractures of the bladder neck. As in the adult, these are best treated by transurethral resection.

Bladder Neck Obstruction Following Operation on the Rectum.

Some degree of dysuria is common after the majority of excisions of the rectum. In Marshall's series it occurred in 50% to 60%. In most cases this subsides after a period of catheter drainage, and parasympatheticomimetic drugs may be of help. In the minority, bladder neck obstruction persists and operation is necessary. Several theories have been put forward to account for the occurrence of the obstruction. Trauma is probably responsible in some of the minor and short-lived cases, but there is never evidence of any severe injury to the prostate or bladder to account for the cases in which operation is required. The neurogenic theory has fallen into disfavour. That injury to the pelvic autonomic nerves is not the cause is shown by the occurrence of the condition after limited perineal resections of the rectum just as frequently as after combined abdomino-perineal operations. Injury to the pudendal nerves could result only from more radical dissection than is normally practised, and the necessary severance of both nerves would produce collateral neurological signs which are not found. The findings on cystoscopic examination are not those of neurogenic bladder. It is generally agreed that the obstruction in many cases is due to preexisting hyperplasia of the prostate, the confinement to bed and operation acting as precipitating factors. On cystoscopic examination, the obstructing prostate is rarely found to be of great size. Resection of the prostate is the method of choice in these cases. Permanent cystostomy is to be avoided in a patient with a colostomy. Suprapubic prostatectomy can be performed, but the disadvantages of this operation are proved by the fact that all these patients are referred to the urologist. Prostatectomy imposes a more exhausting and extensive procedure on a patient recovering from recent major pelvic operation. The presence of a colostomy, often in the left iliac fossa, impedes the surgeon, increases the risk of infection, and presents a major post-operative nursing problem. Those of us who in prostatectomy consider the use of the finger in the rectum a help, are denied this assistance. Since the prostate is usually small, a complete resection is needed for a good result. As judgement of size depends, in these cases, purely on vision and one is denied the aid of palpation, one may be hesitant to cut as boldly as in the normal patient, and further operations may be necessary for cure.

Of a series of six patients in our clinic, all obtained good results, one requiring the removal of further tissue two weeks after his first resection. In some of these cases, perhaps in all, lack of support of the bladder and prostate after removal of the rectum plays a part. Marshall reports that two of his patients became incontinent of urine after resection of the prostate to cure retention of urine. He found that a firm perineal pad caused resumption of normal bladder function. He considers that this state of affairs is analogous to the female incontinence of urine associated with cystocele. In one case he has performed an anterior fixation of the bladder neck, prostatic capsule and anterior bladder wall, with amelioration of the incontinence and a return of bladder function to 90% of normal.

Neurogenic Bladder.

The role of the resectoscope in the treatment of the neurogenic bladder is best discussed under McLellan's main headings.

In the treatment of the uninhibited neurogenic bladder, there is no indication for transurethral operation, the bladder emptying satisfactorily.

In the treatment of the atonic neurogenic bladder, transurethral resection of the bladder neck is not often of value. This is the condition seen in tabes, in pernicious anæmia and other nervous disorders and in diabetes. The neurogenic bladder of diabetes is a relative newcomer to the field, the first resection for its relief having been performed in 1945. It is a condition to be borne in mind in dealing with atypical bladder neck symptoms in diabetics.

The usual finding in the atonic type of neurogenic bladder is funnel-neck deformity or Schramm's phenomenon, to which MacAlpine devotes a chapter in his book. Obviously resection is not needed in the typical case. In some atypical cases, however, especially in tabes, a hyperactive sphincter is present, and resection of this may give some relief even if it is only temporary.

In the third group, classified as reflex neurogenic bladder, due to complete separation of the sacral centre from the cortex as a result of transverse lesions of the cord or extensive brain damage, there may be, in the uncomplicated case, a good useful bladder with little residual urine. In other cases an imbalance between the sphincter and detrusor develops, with incomplete emptying of the bladder. Resection of the hypertrophied bladder neck causes

improvement of bladder function. Whitmore performs the operation in approximately 50% of cases.

Micturition has usually to be initiated and strengthened by contraction of the abdominal muscles and diaphragm or by manual suprapubic compression. The prognosis is much better in patients with incomplete lesions who are ambulatory and whose abdominal walls retain some muscle tone. *In cases in which frequent micturition takes place* as a result of a mass reflex from paralysed legs or other foci, the prognosis is less favourable.

It is in the treatment of the fourth type, the autonomous neurogenic bladder, that resection is of most value. This *is the lesion in which both limbs of the reflex are destroyed.* It results from lesions of the sacral cord, conus or *cauda equina* caused by trauma, *spina bifida*, meningomyelocele *and other lesions. There is no sensation, and reflex micturition* if present is inefficient. There is usually a large amount of residual urine. The urethral sphincter appears *normal or hyperactive, and examination of the detrusor* reveals coarse trabeculation.

The two main methods of treatment are transurethral resection and presacral neurectomy. I have had no experience of the latter operation, but successful results have been reported. *Resection results in improvement in 85%* of cases according to Whitmore, and the relief afforded to the patient makes it a most satisfactory procedure.

In all these cases it is wiser to resect too little than too much, and repeated resections seem to be the usual practice.

Hæmorrhage, at operation or delayed, may be out of all *proportion to the amount of cutting done, and blood transfusion* may be necessary. This feature must be explained by the lack of neurovascular control, the divided vessels remaining wide open.

Bladder Tumours.

The use of the resectoscope in the management of bladder tumours may be discussed under three headings: (i) biopsy *of tumours; (ii) treatment of papillomata of the bladder;* (iii) radical treatment of bladder carcinoma.

Biopsy.

The resectoscope has definite advantages over cystoscopic forceps and loop electrodes. It enables one to remove the selected portion of the tumour with the underlying bladder wall easily, and with control of bleeding. The pathologist can then report accurately on both the morphology and the degree of malignancy of the growth.

Treatment of Papillomata.

In the treatment of papillomata, the use of the resectoscope results in the excision of the whole tumour with a carefully assessed amount of surrounding mucosa and underlying muscle, and the whole specimen is available for microscopic examination and pathological classification. Difficulty is experienced at times in cutting long fronds which tend to float away from the loop when touched. This can be overcome by using the coagulating current to cause the fronds to adhere to the loop, and they may then be divided easily. On the subject of cystoscopic diathermy, MacAlpine states that it is always interesting, three or four weeks after treatment, to discover at cystoscopic examination how much of the papilloma has disappeared. In other words, one does not know exactly how much destruction has resulted, and this procedure is a much less accurate method of treatment. Resection is a more rapid operation than coagulation, and much larger tumours can be removed by this method. The bad practice of multiple sessions is thus avoided, with its difficulty in distinguishing between tumour and the sloughs and œdema of healing bladder wall.

Radical Treatment of Bladder Carcinoma.

The use of the resectoscope as a definite method of treatment of carcinoma of the bladder has been brought forward by Reynolds, Schulte and Hammer. An argument in favour of the use of this method can be propounded along the following lines. The results of surgical treatment of carcinoma of the bladder are unsatisfactory. In a series of *infiltrating carcinomata, Sauer et alii* (1950) found that operation caused prolongation of life for an average of only 2·5 months beyond that of a similar group of untreated *patients. The two major surgical procedures used are* partial and total cystectomy. These operations, as usually performed, differ from most planned operations for carcinoma *elsewhere in the body, in that no attempt is made* to remove the tumour *en bloc* with its efferent lymphatics and lymph glands. Total cystectomy is of value in the less infiltrating tumours of the bladder and relieves the patient of the agony of a "bladder death"; but neither total nor *partial cystectomy is of much greater worth than simple* mastectomy in carcinoma of the breast.

Of 100 cases in which Marshall and Whitmore performed total cystectomy, only 31 were classified as seemingly successful at the end of two years. Some surgeons are per*forming block dissections of the glands, but long-term* survival rates have not been recorded. To my mind, the process of stripping glands from the iliac vessels is flattered *by the term "block dissection".*

Schulte and his associates were so disappointed with *their results from these "open" methods that they have* resorted to transurethral surgery for carcinomata. They use the Thompson resectoscope to remove the protruding *tumour and then the Stern-McCarthy instrument to excise* the affected area of bladder wall. They have abandoned fulguration.

As with resection of the prostate, it will be necessary, over a period of time, to develop a technique in excising *infiltrating tumours adequately. Some tumours will be* difficult to resect, especially those of the anterior bladder wall just inside the bladder neck, and those high up on the posterior wall where the bladder wall is more or less at right angles to the long axis of the instrument, and *hence the excursion of the loop. Some tumours will be too* large to resect. Radical resection will result in intraperitoneal and extraperitoneal extravasation, but these complications have not been frequent, and have not deterred the surgeons.

Jewett and Lewis, in assessing the results of cystectomy, found no cures when the bladder wall was deeply infiltrated, but a high proportion of cures when the tumour extended less than half-way through. This latter degree of infiltration to the mid-point of the bladder wall is well *within the range of endoscopic surgery. Complete removal* of these less infiltrating tumours of better prognosis is thus possible by radical transurethral resection, and there would appear to be no reason why resection should not achieve as many cures as total cystectomy. Schulte and *his associates report that they "have successfully arrested* the growth of multiple infiltrating tumours of both papillary and sessile types in many patients by transurethral resection".

Other Uses.

As well as in the major conditions so far mentioned, the resectoscope has several other uses. It provides an excellent though not the only method of dealing with ureteroceles, and a good method of opening prostatic abscesses.

The fact that the resectoscope sheath possesses as large a bore as any commonly used instrument renders it an ideal sheath for the evacuation of blood clot and fragments of calculus after lithotrity. Occasionally after resection one is confronted after evacuation with a piece of prostate floating in the bladder, too large to be removed by the evacuator. Such pieces can often be engaged by the cutting loop and held firmly against the sheath. The whole instrument is then removed with the piece of tissue impacted in it. This method can also be used with some of those awkward pieces of calculus, too large to be sucked out and too small and flat to be easily crushed smaller. However, loops are ruined by such methods, and they are not recommended for everyday practice. However, with both tissue and calculus fragments I have avoided cystotomy by this means.

The resectoscope may be of value in the treatment of diverticulum of the bladder. After operative relief of bladder neck obstruction a poorly emptying diverticulum may cause persistence of symptoms. Resection or incision of the neck of the diverticulum may improve its drainage, and one may by this means avoid diverticulectomy. Barnes, who coined the term "diverticulotomy" for this procedure, states that with a diverticulum close to the trigone the neck may be incised to a depth of one centimetre with safety. Cutting to this depth is not advisable with diverticula situated higher in the bladder wall.

Summary.

Transurethral resection provides the best method of treatment of bladder neck obstruction occurring in children, in women and in men after excision of the rectum. It is the only helpful surgical procedure in the treatment of many neurogenic bladders. In tumours of the bladder it provides the best method of biopsy and treatment of papillomata, and in infiltrating tumours it may produce results as good as those obtained by any other method.

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Reports of Cases.

INFECTIOUS MONONUCLEOSIS IN AN INFANT AGED TEN WEEKS, PRESENTING WITH JAUNDICE.

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INFECTIOUS MONONUCLEOSIS is a disease noted for the variety of its manifestations, and the case below is reported because of the unusual manner of its presentation, and because of the apparent rarity of the disease at this early age.

Clinical Record.

X, a male infant, aged ten weeks, was referred to Prince Henry's Hospital on February 11, 1954, for investigation, with the following history. The patient was a normal full-term baby (birth weight eight pounds ten ounces), and pregnancy had been uneventful. The mother stated that she had not been ill and had received two injections before delivery. One of these was for routine blood examination at seven months and the other during confinement. The baby had been breast fed whilst in hospital, and thereafter, for no very clear indication, had been placed by the mother on four-hourly feedings of sweetened condensed milk. From the commencement of the artificial diet the baby had regurgitated a considerable portion of each feeding immediately after it was taken. When the baby was aged eight weeks the baby health centre changed the diet to a "Lactogen" mixture, but the regurgitation continued. The mother consulted her general practitioner, who noted that the baby was under weight and sallow, and had an icteric tinge in the conjunctivæ. The baby was therefore referred to hospital for investigation. At this time also the infant's brother, aged three years, was said to be "off colour", but recovered within a day or two without medical attention.

On his admission to hospital, the patient was a thin, lethargic baby weighing nine and a half pounds. There were no developmental abnormalities. The skin was sallow, and the tissue tension was only fair. The conjunctivæ were considered to be of normal colour. The rectal temperature was 99·6° F. The liver was palpable to the extent of one finger's breadth below the right costal margin, the spleen was not palpable, and no other mass or viscus was defined in the abdomen. No lymph glands were palpable in the neck, axillæ or groins. Clinical examination of the heart, lungs and central nervous system gave results within normal limits. The urine contained no bile pigments or bile salts, and a normal amount of urobilinogen. The stools were paler than normal, although containing obvious pigment.

The baby was regarded as a problem of "failure to thrive". Initially he was placed on four-hourly feedings of a "Lactogen" mixture, but the regurgitation continued, so smaller three-hourly feedings were instituted, and with attention to bringing up wind, no further feeding troubles were encountered during the course of the illness.

On the baby's fourth day in hospital it was noted that the urine was dark in colour, and on analysis it was found to contain bile pigments and salts, and no excess urobilinogen. The next day the temperature rose to 104° F. and the child was frankly jaundiced. Besides bile pigments and salts, the urine was found to contain seven polymorphonuclear cells per high-power field in the uncentrifuged specimen, and culture yielded a growth of *Bacillus coli*. A diagnosis of "pyelitis with toxic jaundice" was entertained, and the baby was started on a course of sulphadimidine, and later aureomycin, without noticeable effect on the clinical progress, except that the urine became sterile.

The Pathology Department, under the direction of Dr. John Funder, reported the results of a full blood examination made soon after the child's admission to hospital, and also of an examination made on the day of the onset of jaundice. These are shown in Table I.

The report on the blood film on the child's admission to hospital stated that the red cells were normochromic and normocytic. There were a lymphocytosis and an increase of mononuclear cells, some of which resembled "glandular fever cells". A similar report on the second film carried the following additional remarks: "Although the red blood corpuscles are normal in appearance, the blood picture could be due to hæmolysis".

Coombs's test produced a negative result. Since the hæmoglobin value at the time of the child's admission to hospital was the lowest during the course of the illness, and since the level continued to rise during the development of the jaundice, it was considered that hæmolysis was excluded as a cause of the jaundice.

A Paul-Bunnell test produced a positive result to a titre of 1 in 128. The diagnosis was therefore "infectious mononucleosis with hepatitis".

Tests indicating liver function gave the following results: the serum bilirubin value was five milligrammes per 100 millilitres, the alkaline phosphatase content was 36 units, the total protein content was 6·3 grammes *per centum*, and the albumin-globulin ratio was 2:1; the cephalin flocculation test produced a negative result after

twenty-four and forty-eight hours, and the Wassermann and Kahn tests produced negative results.

The baby's feedings were changed to "Bengerized" skimmed milk, with the addition of vitamin *B* complex, folic acid and "Colliron". These feedings were taken avidly, in quantities of up to 40 ounces per day.

The episode of fever and jaundice lasted with varying intensity for ten days, abated for five days, then recurred for three days. After this the baby was apparently well for two weeks, although there was no gain in weight. Following this there was a febrile period of four days without jaundice, but with recurrence of the urinary infection. This cleared up with a further course of aureomycin, and the baby then became much more active and began to gain weight.

He was discharged from hospital seven weeks after his admission, in apparent good health, and weighing ten pounds five ounces. A blood examination on his discharge revealed a persistence of a mild lymphocytosis, although no abnormal cells were seen on the blood film.

TABLE I.

Observation.	On Patient's Admission to Hospital.	At the Onset of Jaundice.
Hæmoglobin value	9·9 grammes *per centum*	10·7 grammes *per centum*
Total leucocytes per cubic millimetre	14,000	31,000
Differential leucocyte count (per cubic millimetre):		
Neutrophile cells	1,230 (23%)	20,480 (58%)
Eosinophile cells	0	320 (1%)
Basophile cells	140 (1%)	0
Lymphocytes	8,180 (58%)	8,190 (26%)
Monocytes	1,550 (11%)	2,520 (8%)

The baby has since been examined in the out-patient department, and the mother reports continued good health and a weight gain of a pound a week.

Comment.

This case is reported because it presents two unusual features.

The first is that jaundice was an early sign of infectious mononucleosis. Recently, much attention has been focused on the high incidence of liver damage in cases of infectious mononucleosis, the figures ranging from 90% to 100% in various series (Bennett *et alii*, 1950; Watson *et alii*, 1951; De Marsh and Alt, 1947). It is stated (Cohn and Lidman, 1946) that "there appears to be a rough correlation between the severity of the disease and the degree of hepatic impairment".

Observation of clinical jaundice in infectious mononucleosis is a much less frequent event, being quoted variously in between 4% and 10% of cases, whilst even more infrequently is jaundice the presenting symptom. Read and Helwig (1945), in a review of 300 army cases, reported six cases in which the leading symptom on admission to hospital was jaundice.

In infectious mononucleosis, jaundice is regarded as due to true hepatitis, the liver lesion being "essentially a periportal hepatitis . . . hardly distinguishable from liver changes in milder cases of epidemic hepatitis as observed at biopsy" (Custer and Smith, 1948). The differentiation of the two conditions depends on the Paul-Bunnell test.

Most authors stress the value of the thymol turbidity test or of the cephalin flocculation test in demonstrating the presence of liver involvement, and in this regard it is interesting to note that our single estimation of the cephalin flocculation in the case reported gave a negative finding. Indeed, the liver function tests indicate that the jaundice was due to incomplete obstruction of the biliary passages. A liver biopsy would have been desirable, but in such a sick infant investigations were restricted.

We believe, however, that on the weight of evidence from the various series mentioned above, and on the clinical progress of this individual case, the essential lesion in the liver was cellular damage rather than an obstruction to the common bile duct by glands at the *porta hepatis*. A point to be noted was the prolonged period in which the infant failed to gain weight, despite liberal feedings of predigested, high protein diet. This inability to metabolize protein may have been due to liver damage.

Secondly, there is the unusually early age of the patient, for few patients of this age have been reported. Of the 13 patients with infectious mononucleosis at Prince Henry's Hospital in the last four years, the youngest was aged seventeen months. At the Royal Children's Hospital, Melbourne, of the 79 patients listed in the in-patient records in the last ten years, there were four patients aged under twelve months—three of ten months and one of six months. The last-mentioned patient is interesting, in that the child was admitted to hospital with congenital urethral fistula and urinary tract infection, and was found to have an enlarged liver and spleen. Investigation revealed abnormal monocytes in the peripheral blood and a positive response to the Paul-Bunnell test. The infant had had an upper respiratory tract infection three months before admission to hospital.

The case reported and the instance given of the infant aged six months at the Royal Children's Hospital lead one to wonder how many infants suffering from a throat infection with the presence of enlarged cervical glands would, if investigated, prove to be suffering from infectious mononucleosis. Since there is a high incidence of liver involvement in this disease, it is important that these patients be correctly diagnosed and treated with adequate rest and suitable diet. By that means it is possible that another cause may be found and corrected in the big group of infants regarded as suffering from "failure to thrive" after a throat infection.

Summary.

A case is reported of an infant, aged ten weeks, who presented to hospital as a problem of "failure to thrive" and became jaundiced. The diagnosis of infectious mononucleosis was made on the blood film and confirmed by the Paul-Bunnell test.

Comment is made on the incidence of liver involvement in infectious mononucleosis, and on the unusually early age of the patient. The opinion is expressed that the disease may be more common in infancy than is supposed, and that its concomitant liver damage may account for some cases of "failure to thrive".

Acknowledgements.

My thanks are due to Dr. Stanley Williams, Honorary Pædiatrician to In-Patients, Prince Henry's Hospital, and to the Medical Superintendent of Prince Henry's Hospital, for permission to report this case; to the pathology department, under Dr. John Funder, for the laboratory investigations; and to Dr. Donald Forster for the hæmatological reports. The Medical Director of the Royal Children's Hospital, Melbourne, kindly allowed me to consult, and to quote from, the records of that hospital.

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Reviews.

Seventy-Five Years of Medical Progress, 1878-1953. Edited and with a foreword by Louis H. Bauer, M.D., F.A.C.P.; 1954. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 9½" × 6½", pp. 286, with 25 illustrations. Price: 43s.

THIS book is made up of 20 papers read at the first Western Hemisphere Conference of The World Medical Association, which was held in Richmond, Virginia, in April, 1953. The theme of the conference was the commemoration of seventy-five years of medical progress. Distinguished representatives of 19 medical specialties, as well as a representative of general practice, contributed papers on the history and the present status of their particular fields in medicine. Although a good deal of information is packed into a relatively small space, the authors of the several papers have contrived to make their papers readable, and they should retain their interest and value permanently as a record of contemporary medical history in a remarkable medical era.

CIBA Foundation Symposium on the Kidney: Arranged Jointly with the Renal Association. Editor for the Renal Association, A. A. G. Lewis, B.Sc., M.D., M.R.C.P.; editor for the CIBA Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8" × 5½", pp. 350, with 125 illustrations. Price: 32s.

THIS Ciba Foundation volume is a verbatim account of an international symposium on the kidney, arranged jointly by the Ciba Foundation and the Renal Association and held in London in July, 1953. There were 35 participants from Great Britain, Ireland, the United States, Sweden, Holland, Belgium, Switzerland and France, all experts on renal physiology, pathology or medicine. There were five sections as follows: structural and functional relationships in the kidney, with four papers, all with discussions; tubular functions other than the regulation of acid-base balance, with four papers and discussions; renal share in the regulation of acid-base balance, with four papers and discussions; general problems of electrolyte excretion with five papers and discussions; and renal share in volume control of body fluid, with three papers. The ground traversed in the 22 papers is very extensive, covering many features of renal excretion. It is not possible to discuss all the papers, but a few may be mentioned. A paper on preliminary experiences with aspiration biopsy of the kidney by F. Raaschou deals with a new technique for obtaining specimens from the living kidney for histological examination. The photographs of sections show excellent detail. J. V. Taggart discusses the biochemical mechanisms of various renal activities in some biochemical features of tubular transport mechanisms. A paper on renal response to massive alkali loading in the human subject by P. H. Sanderson throws some new light on alkalosis. N. Alwall deals with ultra-filtration and dialysis of blood *in vivo* in the treatment of electrolyte-fluid retention. The papers on electrolyte excretion and retention are particularly good. Not the least interesting are the discussions, which at times were very spirited. The book can be recommended to anyone interested in the kidney and its disorders.

Pediatric Problems in Clinical Practice: Special Medical and Psychological Aspects. Edited by H. Michael-Smith, Ph.D., with fourteen contributors; 1954. New York: Grune and Stratton, Incorporated. 8" × 5½", pp. 320, with four text figures. Price: $5.50.

THIS is an addition to the growing list of books which are really a collection of contributions by leaders in their own branch of medicine, the whole being edited by one of the authors. This volume is the work of 14 writers, and contains sections on practically every aspect of child health and disease that the general practitioner and the pædiatric specialist sees. The sections on the normal child and the generally sick child are treated in a less satisfying manner than some of the other sections—for example, those dealing with the emotionally disturbed child, the allergic child and the cardiac child. Other sections are devoted to the schizophrenic child, the mentally retarded child, the brain-injured child, the cerebral palsy child, the orthopædically handicapped child, the diabetic child, the epileptic child and the tuberculous child.

The manner of treatment of each section is fairly uniform; each author discusses the causes of the condition, its effect upon the whole child and the family, and the general lines of treatment. In view of the relatively large number of children who exhibit some degree of emotional disturbance, the chapter on this subject should prove particularly useful. Although the author has obviously drawn heavily upon the current ideas about child development, including contributions from analytical theory, his presentation is made in a straightforward manner; explanations are given of how the deviations at each stage of development may lead to emotional problems. A pleasing feature of this book is the extent to which the modern outlook of considering the whole child in relation to the disease or condition for which the child is under the care of the physician is incorporated into each section. Although the book is orientated towards the emotional aspects of each clinical condition, a nice balance is maintained between the emotional and physical factors in the discussions on ætiology. This is particularly noticeable in the section devoted to the allergic child.

This book should prove of value to the general practitioner who is anxious to keep abreast of latest concepts about the management of children suffering from one of the diseases or handicaps covered by it.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Ciba Foundation Symposium on Hypertension: Humoral and Neurogenic Factors." Editors for the Ciba Foundation, G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., assisted by Joan Etherington; 1954. London: J. and A. Churchill, Limited. 8" × 5½", pp. 308, with 73 illustrations. Price: 30s.

The symposium comprises twenty-one contributions with a discussion on most of them.

"Demonstrations of Operative Surgery: A Manual for General Practitioners, Medical Students and Nurses", by Hamilton Bailey, F.R.C.S., F.A.C.S., F.I.C.S., F.R.S.E.; Second edition; 1954. Edinburgh and London: E. and S. Livingstone Limited. 9" × 6", pp. 400, with 538 illustrations, a few in colour. Price: 24s.

Originally designed for nurses, but now extended for the benefit of medical students and general practitioners.

"The Foundations of Surgery", by George Perkins, M.C., M.Ch., F.R.C.S.; 1954. Edinburgh and London: E. and S. Livingstone Limited. 7½" × 5", pp. 244. Price: 10s.

Intended for reading by the medical student in the first few months of his clinical training period.

"Anatomy: Regional and Applied", by R. J. Last, M.B., B.S. (Adelaide), F.R.C.S.; 1954. London: J. and A. Churchill Limited. 10" × 7", pp. 678, with 309 illustrations, some in colour. Price: 55s.

Written in the light of an extensive experience with postgraduate students of anatomy.

"Emergency Treatment and Management", by Thos. Flint, junior, M.D.; 1954. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Limited. 9½" × 6½", pp. 314. Price: £2 14s. 9d.

Aims to present the medical and surgical management of the patient with an emergency condition from first examination until disposition for definitive treatment can be arranged.

"Biochemical Investigations in Diagnosis and Treatment", by John D. N. Nabarro, M.D., M.R.C.P.; 1954. London: H. K. Lewis and Company Limited. 8½" × 6", pp. 308, with five illustrations. Price: 25s.

Written as a practical guide for hospital residents and registrars.

"Proceedings of the Fourth International Congress of the International Society of Hematology: Mar del Plata, Argentina—September 20-27, 1952", associate editors, F. Jimenez de Asua, William Dameshek and Sol Haberman; advisory editors, Jean Bernard, O. P. Jones, P. Introzzi, Sven Moeschlin and L. M. Tocantins; assistant editors, Gonzalo E. Aponte and Jorge Lajous; 1954. New York: Grune and Stratton. 10" × 7", pp. 486, with 232 illustrations. Price: $10.00.

A report of the deliberations of over 300 hæmatologists.

The Medical Journal of Australia

SATURDAY, NOVEMBER 13, 1954.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE FLUORIDATION OF WATER AND DENTAL CARIES.

REPEATED reference has been made in these columns to the fluoridation of water as a means for the prevention of dental caries. The subject has been discussed at several meetings of the Federal Council and it has also been before the National Health and Medical Research Council. In October of last year it was discussed at considerable length in these columns and attention was drawn to a report issued by the Ministry of Health of Great Britain. The report was that of a United Kingdom mission to the United States to inquire into the value of the measure. Readers may remember that the main conclusion of that report was that epidemiological studies in America had shown beyond doubt that among children and adults who had been brought up in areas where the drinking water contained fluorine at a level of one part per million or more there was much less dental caries than in areas where the water was free from fluorine. It was stated that the benefit derived by young children from the use of fluoridated water accompanied them into adult life. The United Kingdom Mission could find no sound evidence demonstrating any ill effects of fluoridation, though it admitted that there was some genuine scientific disagreement in America about the subject. The subject was dealt with at some length by the National Health and Medical Research Council at its thirty-sixth session held at Canberra on December 4, 1953. The Federal Council's representative on the National Health and Medical Research Council, Dr. W. F. Simmons, in his report to the Federal Council set out the resolutions adopted by the National Health and Medical Research Council dealing with the matter. The Federal Council, at its meeting in March, 1953, had Dr. Simmons's report before it and expressed general agreement with the findings set out in the resolution, and it determined that it would give what support it could to them. The resolutions of the National Health and Medical Research Council dealing with this matter have not been published up till the present time, when they appear in the official report of the December meeting of the Council. It is thought that a résumé of the resolutions should be set out so that medical practitioners will have some facts on which to base their views when, as is quite likely, the matter is brought before them by non-medical authorities.

The resolutions of the National Health and Medical Research Council that must be referred to are two in number. The second must be dealt with first because Resolution 1 is conditional upon it. The second resolution states the recommendation of the Council that each State should set up an advisory panel within its health department to review, advise on, and supervise proposals for the addition of fluorine to communal water supplies. It was thought that these panels should include representatives of the medical and dental professions and of public health, and also a water engineer and a chemist. Emphasis should be placed on the fact that these advisory panels are "to review, advise upon and supervise". This means that the opinions set out at length in the first resolution are not to be regarded as the basis for action. As a matter of fact, the National Health and Medical Research Council has shown justifiable caution in the way in which it has handled this matter. The first resolution opens with an expression of opinion that an optimal intake of fluorine is a factor in the prevention of dental caries. That conclusions should not be hurriedly drawn from this is obvious from the addition to the clause which reads that it has to be admitted that an adequate supply of fluorine in drinking water will not, of itself, provide the solution of the problem of dental caries—there are other factors, particularly dietary factors, involved in its control. The Council then adds that, having considered published reports of the claims for and against artificial fluoridation of public drinking supplies, it recommends that when the optimal intake is not obtained from natural sources, fluorine should be added to the public water supply. It is explained that the habitual use from early infancy onwards in a temperate climate of water containing one part per million of fluoride has been shown to confer the greatest degree of freedom from dental caries which can be secured by this means without risk of disturbance of any bodily structure or function. Here we must take notice of the implication that if the intake is not optimal there may be disturbance of a bodily structure or function. The resolution states that for such children, the average daily intake of fluoride from all sources when the water contains one part per million of fluoride has been determined for various age groups from one to twelve years. According to age, this intake ranges from 0·4 to 1·7 milligrammes. For adults, an average daily intake of water ranging from 1200 to 1500 millilitres would result in an intake of 1·2 to 1·5 milligrammes of fluoride. In addition, there would be a computed daily intake of 0·2 to 0·3 milligramme of fluoride from food, the total daily intake of fluoride being, there-

fore, 1·4 to 1·8 milligrammes. It is further stated that on the basis of observations made on a population whose daily fluoride intake is within the range stated and from observations on the excretion of fluoride, there is no evidence that fluoride will accumulate in the body to an undesirable extent when the daily intake is less than three milligrammes of fluoride. The definite statement is made that there is no conclusive evidence that any deleterious systemic effects will follow the habitual use of water containing one part per million of fluoride. So far, the resolution deals with quantities of fluoride which must be added to water, and this, it must be insisted again, is of basic importance. Even the statements already made are not the kind of statements which can be accepted and acted on without further reference to the future. Another clause of the resolution states that although the Council can see no reason why the dental benefits of fluoridation of water should at the present stage be denied to the Australian people, it must be emphasized that concurrent research is essential in order to assess the results of treatment of the water and to determine accurately the optimal concentration of fluorine under Australian conditions. The conditions under which any plan to fluoridate domestic water supplies must operate are set out as follows: (*a*) The need for increasing the concentration of fluorine in the water supply must be established. (*b*) A large proportion of the community should desire fluorine to be added to the water supply, or alternatively, a substantial proportion of the community does not oppose the addition of fluorine to the water. (*c*) The water supply must be amenable and subject to strict supervision and control by qualified engineers and chemists. (*d*) The amount of fluorine to be added must be carefully determined and adjusted to meet climatic and environmental changes. The Council thought that a properly controlled national study of water fluoridation under Australian conditions should be instituted immediately with three objects—first of all, to determine the results of water treatment; secondly, to determine more accurately the optimal concentration of fluorine; thirdly, to permit of valid comparisons between the observations made in different parts of Australia. It was thought that the study should embrace: (*a*) a survey of oral conditions in representative samples of children and adolescents, (*b*) the influence of climate upon water consumption, (*c*) the excretion of fluorine in the urine of sample groups of children and adults. In order that comparable results might be obtained, the Council recommended the establishment (at the Institute of Dental Research, under the supervision of Dr. N. E. Goldsworthy) of a course of instruction in standardized procedures and practice for oral examination. In order to coordinate the study of the influence of climate upon water consumption and the excretion of fluorine in urine, the Council recommended the appointment of an advisory panel consisting of Professor Macfarlane, Dr. Eben Hipsley, Dr. N. Crosby and Dr. N. E. Goldsworthy. The Council considered the question of self-medication with fluoride and strongly deprecated inexpert and indiscriminate self-medication for the purpose of partial control of dental caries. The conclusion to be drawn from all we know about this subject is that fluorine is a useful weapon against dental caries, but that its use demands unceasing supervision and continuous research.

Current Comment.

THE HEART OF ROBERT BURNS.

ON January 25, 1759, near Alloway Kirk in Ayrshire, was born Robert Burns, who has been called "the Shakespeare of Scotland". He was the first child of William Burnes[1] and his wife, Agnes Broun. Agnes, vivacious, red-haired, brown-eyed, and with a retentive memory for the songs and ballads of her country, could read but not write. Of William Burnes, Thomas Carlyle wrote the following words:

> The poet was fortunate in his father—a man of thoughtful, intense character . . . valuing knowledge, possessing some and open-minded for more, of keen insight and devout heart, friendly and fearless. . . . But poverty sank the whole family even below the reach of our cheap school system, and Burns remained a hard-worked plough-boy.

Esther H. Vincent has written an interesting account of Burns's life with particular stress on the medical aspect.[2] She describes his childhood and adolescence, during which were sown the seeds of the disability that ultimately brought about his death. William Burnes gave to his seven children such education as he could afford; when he could afford none, he taught them himself by candle-light, "trying to give them not only love of learning but something of his own religious faith and courage". From Robert's seventh year the family struggled hard to gain a living from rented farms. During eleven years spent on the first farm, at Mount Oliphant, the boy had many bouts of nervous depression and dull headaches at night; later he suffered from cardiac palpitation with feelings of faintness and suffocation. When he was thirteen years old he helped in the threshing of the grain, and at fifteen years he was the chief labourer on the farm. William Burnes had not married young, and was now feeling the effects of hard work and poverty; he and his two eldest sons, Robert and Gilbert, were hard put to it to wrest a living from the farm. Esther Vincent believes that Robert's later physical and nervous illness can be largely traced to this period. The next farm was near Tarbolton, where Robert enjoyed the social life; the family moved there when he was eighteen years old, and he lived there until he was twenty-two. Then he tried a flax-dressing business at Irvine, and while he was there he had the first of the several nervous breakdowns from which he suffered. He had much personal magnetism, inherited from his mother; but his father had endowed him with a tendency to melancholy, as well as tenacity of purpose. In his own words, "the least anxiety or perturbation in my breast produces the most unhappy effect on my whole frame". The business venture was a failure (his partner was a rogue and the shop was burned), and Robert returned to the family farm. There disaster faced the family. The American Revolution had thoroughly upset the Scottish economy, and bad weather ruined the crops. William Burnes, completely worn out, died in the following winter from "a phthisical consumption". Robert and Gilbert rented another farm, Mossgiel, which proved to be just as unrewarding as their previous adventures in agriculture. In 1874 Robert had a severe physical breakdown; Esther Vincent believes that it may have been an early manifestation of a recurrent heart condition.

In spite of his tendency to melancholy, Burns was a most convivial person and highly susceptible to "the lassies", who in turn were equally susceptible to his charms. His physician at this time was Dr. John Mackenzie, a man of his own age and a good friend, who helped him along the literary path which took him to Edinburgh. "Nevertheless, he knew little of heart disease, and in his way played as much havoc with Robbie's heart as did the bonnie lassies in theirs." His treatment included cold baths and continued farm work; in spite of this Burns recovered from the attack with its associated "hypo-

[1] The name was originally Burness; Robert used the form "Burns" to suit the Ayrshire pronunciation.

[2] *Surg., Gynec. & Obst.*, August, 1954.

chondria", but some damage remained. We need not concern ourselves in this place with his literary activities; but it is interesting to note that the list of subscribers to the first edition of his poems contains the names of several medical practitioners—Mr. John Bell, surgeon, Dr. Henry Cullen, Dr. Andrew Duncan, Dr. James Gregory, Dr. Alexander Monro, Mr. John Rae, surgeon, and Dr. Alexander Wood. The "unlettered plowboy" went to live in Edinburgh, and was accepted and lionized. In spite of his recurring disabilities he was a strong, robust man, with an attractive appearance and personality. He made a pen-friendship with Dr. John Moore, the father of Sir John Moore, of Corunna fame, who lived in Glasgow; Burns and Dr. Moore never met. Another companion was Dr. James M'Kittrick Adair. Burns returned to Edinburgh for a second winter and lived more quietly. He suffered from what he called his "bitter blue-devilism", from trouble with publishers, and from the effects of being thrown from a coach driven by a drunken driver. This accident severely damaged one of his knees, which never properly recovered; indeed, H. B. Anderson, who wrote an account of Burns's life, considered that the injury localized to that one joint the rheumatism from which he periodically suffered, and so was the beginning of his crippling arthritis. Burns had many love affairs, and Esther Vincent's words on the subject deserve quotation:

> . . . he valiantly battered himself into warm affection for any girl who loved him. But he was an imperfect Don Juan, for he lacked the sinister courage to desert the victim, and was consequently perpetually entangled. Furthermore, he tenderly loved and cared for those of his children whose mothers would part with them.

It is pointed out that his affairs were always with "socially acceptable lassies who took the initiative in the wooing", and that there is not a shred of evidence that he ever suffered from "the accidental complaint". His wife, Jean Armour, never showed any sign of infection; and the fact that Burns himself did some of his best work near the end of his life does not suggest that disease had affected his ability.

Realizing that his popularity in Edinburgh must ultimately wane, Burns rented another farm, Ellisland, applied for a position as exciseman, and married Jean Armour. Ellisland was as unfortunate a proposition as the other farms, and in addition it had an "atmosphere of fevers and rheumatism". There Burns laboured hard, and rode about in all kinds of weather carrying out his work as a gauger. He had hardly ever been completely well since the days at Mount Oliphant, having suffered from frequent attacks of fever, colds and severe "hypochondria". Now everything was very much worse, and financial worries caused him nervous headaches as well. In 1791 he fell with his horse, sustaining injuries to his hand and arm, later fractured his arm, and later still was incapacitated by a severely bruised leg. The family left Ellisland, and moved to Dumfries. There were then three living children, all aged under six years, and they were joined by Burns's two illegitimate daughters, who grew up as members of the family. In 1792 another daughter was born to Robert and Jean; she was always frail, and died in 1795, having caused her loving father much anxiety all her life. (Burns's son was born on the day of his father's funeral, and was named Maxwell, after Dr. William Maxwell, his father's last physician and very good friend.) In 1793 Burns's salary as an exciseman made it possible for the family to move to a larger house and live in some comfort. For a while he came out of his depression, enjoyed life (in spite of being obliged to rest a great deal) and wrote some of his best poems. However, in 1794 he almost broke down again; but he recovered and went back to his work as an exciseman, this time as a supervisor. He took an active part in civic affairs; but late in 1795 he fell seriously ill with fever, which waxed and waned until his death on July 21, 1796. His arthritis was called "flying gout" by his medical attendants. Early in July, Dr. Maxwell recommended sea bathing and country life, so the unfortunate patient heroically obeyed orders. He came home, much worse, felt that his end was near, and wrote to Jean's mother, asking her to come and look after her daughter. Three days later he was dead.

Esther Vincent refers to the biography of Burns by Dr. James Currie, who was sincerely anxious to help his widow and children; however, Currie depended on such information as he could obtain from Burns's friends, and managed to gain the impression that he was an alcoholic (Currie himself was a fanatical temperance reformer). There is plenty of evidence to disprove this misapprehension, and it is well that the harm done unwittingly by Currie has been put right. It is quite clear that Burns drank rather less than his contemporaries. Sir James Crichton-Browne holds that Burns died of rheumatic endocarditis. H. B. Anderson refers to his rheumatism with heart complications, and considers that bacterial endocarditis developed as a terminal infection. Anderson points out that Burns was a person of physical and mental strength with an intense, nervous temperament and given to rapidly varying moods; in such a person it is not unusual for stress and emotional disturbance to produce the characteristic symptoms of headache, sleeplessness, digestive disturbance, irritability, apprehension, depression and so forth. The relationship between Burns's various breakdowns and the hard work, privation and exposure of his adolescence is obvious; these were predisposing factors to the rheumatism and endocarditis which afflicted him. S. Watson-Smith remarks that painful arthritis and terminal anxiety and dreadful suspense are not uncommon complications of Burns's type of illness.

This brief résumé of the life of Scotland's greatest poet deals almost entirely with one aspect, his health, and chiefly with one aspect of that, his heart. Nothing has been said of his literary achievements, and nothing need be said. But two lines from his *Epistle to Davie* may be quoted, since they almost epitomize himself:

> The heart ay's the part ay
> That makes us right or wrang.

ON THE INTERNAL ADMINISTRATION OF HOSPITALS.

ON March 14, 1950, the Committee on the Internal Administration of Hospitals was appointed by the Central Health Services Council to inquire into the internal administration of hospitals. The report of this committee has been issued by the Ministry of Health of Great Britain.[1] The Committee on the Internal Administration of Hospitals consisted of 20 persons. Of these, ten were members of the Central Health Services Council and three of them had medical qualifications. There were ten members of the Committee who were not members of the Council, and three of these had medical qualifications. The terms of reference of the Committee are set out as follows:

> To consider and report on the existing methods of administration in individual hospitals *and within Hospital Management Committee groups*, with particular reference to: (i) Matters of finance, staff and supplies; (ii) the extent to which differences in the work undertaken at different hospitals call for differences in their administrative organization; (iii) the extent to which administrative duties should be undertaken by medical and nursing staff.

The use of italics in these terms of reference is of interest. The italicized words were not in the original terms, but were added at the request of the Committee because it was thought that without these words the Committee would not be able to investigate what were emerging as the most difficult and important questions in hospital administration.

It will be clear to Australian readers that this report will not be entirely applicable to Australian conditions because of the advent of the National Health Service in Great Britain. The Committee points out that the subjects investigated by it are by no means new, but explains that the grouping system of hospitals has complicated the question.

[1] Report of the Committee on the Internal Administration of Hospitals; Ministry of Health, Central Health Services Council: 1954. London: Her Majesty's Stationery Office. 9½" × 6", pp. 87. Price: 3s. net.

The Committee tried to approach the task with the idea in mind that throughout the service the administrative pattern should remain flexible, so that it would fit not only the different circumstances of different groups, but also the different circumstances of the same group at different times. The Committee held, in all, 24 meetings. It sent out to a large number of representative bodies a *questionnaire* dealing with seven matters. In our account of the matters set out in this report, mention will be made of only the more important items.

In the section dealing with the administrative structure of the hospital group it is stated that the primary function of the governing body (this term is used throughout the report to indicate both hospital management committees and boards of governors) is to ensure that all the efforts and skill of the management and staff are employed efficiently, quickly, and with due economy in the interests of the patients. Hospital administration is described as a tripartite administration, and is divided into: (*a*) medical administration; (*b*) nursing administration; (*c*) lay, or business, administration. It is stated that the conception of partnership between the three parts of the whole administration should be regarded as fundamental and should determine the lines of all future development, whatever variations of superficial pattern may be necessary to give expression to varied circumstances. "Partnership" is regarded as the important word. None of the three parts can do without the others; each has its own functions which it is better adapted to perform than the others, and none should regard itself as superior to the others. Cooperation alone is not enough; it must be willing cooperation, springing from a consciousness of fellowship in a shared desire to serve. Three general principles are stated as follows: (i) All administration must always be carried out within the general policy of the governing body. (ii) Because of the inevitable interaction of their duties, all who are doing administrative work must work together as a blended team. (iii) Neither a medical man nor a nurse should, as a rule, do work which a layman can do equally well. We should imagine that the third of these principles has been laid down because of a shortage of trained medical administrators. Experience in this country has shown that when the administrator of a hospital, trained in administration, has a medical qualification as well, his work is likely to be more effective, and efficiency is better served than it would be if he was a layman. The governing body of hospital groups should have one officer to whom it can look for securing that its policy is carried out in all hospitals in the group, and for coordinating and reviewing all group activity. It is stated that the Committee regards it as essential to develop a genuinely tripartite administrative organization at the individual hospital under the person responsible for its administration. It is impossible at a group level to visualize any such clearcut division of function expressed in terms of individual officers.

Interest will probably centre about what has been written in regard to the administration of general hospitals. Here, we note that more than half the written evidence submitted to the Committee, and a proportionate time spent in discussion with witnesses, was concerned with the question of medical versus lay administration: not the proper sphere of each and their proper relationship and interaction, but the two spheres considered over against one another as mutually exclusive. This was unfortunate. Five possible forms of medical administration are set out as follows:

(1) Medical superintendent with direct control over matters of medical administration and oversight of all hospital departments, but with clinical responsibility confined to his own patients.

(2) Medical administrator with functions limited to medical matters and without authority over the field of lay administration.

(3) Physician or surgeon superintendent who, in addition to clinical duties, has certain part-time administrative functions as under heading (2).

(4) Chairman of medical staff committee with a resident medical officer of less than consultant status concerned with admissions only.

(5) Chairman, or a member of the medical staff committee, without resident medical officers, all day-to-day administrative matters being handled by individual firms.

It is pointed out that under headings (2) to (5) inclusive there must be a medical staff committee; such a committee is not necessary under heading (1). Several arguments in favour of medical superintendence are set out. The first is that a hospital is an institution providing a medical service, and "its head should, therefore, be a medical man, as being best equipped to distribute its resources and to ensure by his understanding of medical considerations, for which the layman has not had the training, that a comprehensive medical service is provided and that the hospital does not become an association of departments without integration or central purpose". The second is that only a medical man can perform certain administrative duties in a hospital. The duties ascribed to the Committee have usually included such functions as the reviewing of the waiting lists of patients for admission in all departments, and relations with general practitioners and the various senior technicians of the hospital. The third argument is that laymen lack both fundamental knowledge of the patient's needs and authority with the medical and nursing staff. No amount of training in hospital administration can, it is stated, remedy these deficiencies. The arguments against medical superintendence are partly lay and partly professional—the former, stressing the medical man's lack of administrative experience and the extremely complex nature of the non-medical problems facing the administrator, the latter, the danger of clinical dictation and the loss to clinical work, often entailed by the employment of a trained consultant on administrative work. These views were put to the Committee by a variety of bodies. Under the heading of medical administration in general non-teaching hospitals, the functions of a medical administrator are stated to include the following:

(1) Continuous advice to the governing body on the best use of the available beds and all other facilities for patients;

(2) co-ordination of the work of medical departments and matters of medical administration arising therefrom;

(3) liaison work on medical matters with the Regional Board, the Local Health Authority, the local Executive Council, the Coroner, etc.;

(4) medical supervision and control of admissions and discharges;

(5) the survey of waiting lists and the action to be taken to meet varying pressures on different departments;

(6) departmental supervision and co-ordination of medical auxiliaries;

(7) medical aspects of the almoner's work;

(8) the medical arrangements to cover the smooth-running of the out-patient clinics, appointments system, casualty department, etc.;

(9) supervision of medical equipment and medical supplies, in co-operation with the chief pharmacist;

(10) medical records;

(11) hospital discipline involving patients;

(12) discipline of junior medical officers;

(13) general supervision of the health of the staff;

(14) notification of infectious diseases; co-ordination of measures to prevent the spread of infection within the hospital and the investigation and control of any outbreaks of epidemic disease or cross-infection within the hospital, in association with the local authority where necessary;

(15) supervision of any area service provided by the hospital, e.g. obstetric flying squad, blood transfusion service where appropriate.

The conclusion is stated that when the content of a medical administration is examined, it transpires that practically the whole of it, if not directly concerned with the medical treatment of an individual patient, is indispensable for the adequate medical treatment of the patients as a whole. The control of cross-infection in infectious disease, the proper admission and disposal of patients as determined by their own and the community's needs, the

coordination of out-patient clinics and bed allocations in such a way that the patient's needs are satisfied in accordance with medical urgency, attention to the regular maintenance of the records and communication with family doctors and outside agencies, including the local service—all this, and indeed practically the whole of medical administration, require expert clinical knowledge and are clinical in essence. The medical administrator, as conceived by the Committee, should be regarded as the agent of the medical staff committee in all matters of administration, and he can best, probably, carry out his duties if he is their honorary secretary. All this seems somewhat at variance with the conclusion previously stated in regard to hospital groups, that neither a medical man nor a nurse should, as a rule, do work which a layman can do equally well. If the conclusions already stated apply, as they appear to apply, to medical administration in non-teaching hospitals, it is difficult to understand why they should not apply to hospital groups.

The section dealing with nursing administration is full of interest. Full discussion of it should be left to a future occasion; the opinion, however, should be noted that it is undeniable that it is primarily the matron and her nursing staff who create the comfort, the content, and the atmosphere of the hospital, and who can most influence the feelings with which the general public regard it. The relationships of the matron to the governing body of the hospital and of the trained nurses on the staff to the matron are discussed, and some interesting suggestions are made. In regard to lay administration, it is held that certain administrative functions are wholly lay in character. The functions coming under this heading are summarized under the headings of finance, supply, establishment, the maintenance of building and plant, laundry, catering and food supply, farms and gardens, clerical and recording work. Expert knowledge on these matters can of course be acquired by a medical graduate who is willing to study and master them. Under the heading of the "committee system" reference is made to the duties of house committees.

GLUTEN-FREE MANAGEMENT OF CŒLIAC DISEASE.

CŒLIAC DISEASE was first described by Gee in 1888, but it has since been one of the most baffling diagnostic and therapeutic problems in clinical pædiatrics. The disease has never been accurately defined. It may first show itself at any time between later infancy and middle childhood. A healthy child over a period of a few weeks ceases to gain weight, goes off his food and may vomit occasionally; the stools become paler, bulkier, softer and more offensive. The child's weight oscillates widely from day to day; he becomes thin, the abdomen becomes protuberant, the limbs and buttocks become wasted. The condition may persist through childhood with violent crises of dehydration, one of which may be fatal. Gradually the symptoms disappear, leaving a malnourished dwarf. Gee gave his opinion that the cure would be found in dietetic management and that a restriction in the intake of farinaceous food was essential. Later observers believed that intolerance to fats was the principal point to be noted.

D. Lawson describes the progress of the dietetic treatment of the disease initiated by W. Sheldon and continued by Sheldon and Lawson at the Great Ormond Street Hospital since 1948.[1] At first a strictly starch-free diet was given. The fat absorption rose to normal figures, the children lost their symptoms within weeks and rapidly regained normal health. This starch-free diet was, incidentally, also gluten-free. In 1950, Dicke and other workers in Holland reported that pure wheat starch, from which all protein had been removed, could be added with impunity to the polysaccharide-free diet, but that the symptoms of the disease recurred if gluten, the protein component of wheat flour, was added. Sheldon and Lawson reviewed all their cases in which treatment with starch-free diet had been adopted. Fifteen patients out of 64 showed an unsatisfactory nutritional state which improved rapidly on a gluten-free diet. A starch-free diet is very difficult to maintain in the home, and the unsatisfactory condition of these children was no doubt due to slips. When gluten is removed from the diet of patients, there is not an immediate response, and two to six weeks may elapse before improvement is noted. There is now a series of 120 children being studied at Great Ormond Street Hospital after the initiation of a gluten-free diet, and the results fully confirm those reported earlier. The change-over in the diet in the early weeks of treatment of an uncooperative and miserable child is difficult and demands much patience. At this stage the diet has consisted of a dried milk product, prosol, with added sugar. This is gradually increased in amount; then banana, egg custard, lean meat and chicken are added. Gradually a diet of normal constitution, with complete absence of all food containing wheat or rye, is achieved. Flour is replaced by pure wheat starch or cornflour. There are great difficulties here in home treatment, especially of school children. The nature of the toxic substance in gluten is not known, but it is probably a protein. Gluten is an ill-defined mixture of proteins of different properties.

[1] *Proc. Nutrition Soc.*, 1954, Volume 13, Number 2.

BRITISH EMPIRE CANCER CAMPAIGN.

THE thirty-first annual report of the British Empire Cancer Campaign covering the year 1953-1954 maintains the traditions of previous publications, especially as to the large volume of work and the wide geographical distribution of the places where research has been carried out. In some cases the investigations are incomplete but still in progress; negative results are wisely given; though these are rarely spectacular they reduce the unknowns and act as deterrent guides to future research. As in former years, there are contributions to theoretical science such as the chemical effects of radiation, and to technique, for example, electron microscopy, and the application of the flying spot principle and other television-like procedures to microscopy.

Some of the reports present tentative hypotheses which are likely to be true, such as wound hormone leading to epithelial proliferation and the possible importance of blood groups not only in cancer but in non-malignant pathological states; thus peptic ulcer is more common in blood group O. Amongst the more important findings the following may be mentioned. There are co-carcinogens such as croton oil unable themselves to produce cancer but rendering a subliminal dose of carcinogen effective, or actually turning a non-carcinogenic substance such as urethane into an active promoter of cancerous growth. The separation and classification of products of cigarette smoking have been, as might be expected, actively undertaken. So far the verdict on tobacco smoking is that, up to date, the Scots' "not proven" is recommended. A naturally occurring œstrogen, genestein, and certain synthetic substances related to it, have been found to be highly carcinogenic. Two of the alkaloids of the South African ragwort which induce malignant liver tumour have been studied; further investigations of other alkaloids from this plant are being pursued.

Some readers of this voluminous report may be inclined to feel that if liberal scholarships and grants are available for cancer investigation there will always be an abundance of researchers usually following the directions of the several heads of departments where they are working and whose publications sometimes border on the pedestrian; but this is a dangerous attitude and any critic who labels a piece of work as devoid of inspiration or applicability may in the near future be forced to eat his words. It is possible that some earnest readers may not find this report as optimistic as its predecessors, but again caution is necessary; next year's report may display great advances along lines modestly suggested this year.

Abstracts from Medical Literature.

THERAPEUTICS.

Treatment of Cardiac Arrhythmias.

L. R. PASCALE *et alii* (*Am. Heart J.*, July, 1954) have treated 40 paroxysms of tachycardia occurring in 33 patients with intravenous injections of procaine amide and found this drug very successful except when auricular flutter was the cause of the tachycardia. The drug was administered at a maximum rate of 100 milligrammes per minute, but no arbitrary limit was set for the total dose. The electrocardiogram was under continuous observation, that lead being selected which best showed atrial activity. Sometimes the recommended dose of 1000 milligrammes had to be considerably exceeded, serious hypotension being combated with infusions of *l*-noradrenaline. One of the patients suffered convulsions and death, after which as a routine an intramuscular injection of 100 milligrammes of soluble phenobarbitone was given before the treatment.

Hydrocortisone.

T. W. FYLES AND B. ROSE (*Canad. M.A.J.*, June, 1954) discuss the oral administration of compound F for asthma. They state that 17-hydroxycorticosterone or hydrocortisone is excreted in large quantities when ACTH is adminstered. In the same dosage hydrocortisone produced an eosinopenic response twice as great as did cortisone in a patient with Addison's disease. For rheumatoid arthritis, compound F was more effective than cortisone in smaller doses. Fourteen patients with bronchial asthma were treated with hydrocortisone by mouth; 20 milligrammes were given four times daily. The response was prompt and striking in 14 cases, within one to six days. The dose was reduced gradually to a maintenance level of 20 to 80 milligrammes per day. The results were much the same as with cortisone, but the doses were smaller. Side effects were rare even after two or three months.

Cortisone and Rheumatoid Arthritis.

F. W. B. HURLBURT *et alii* (*Canad. M.A.J.*, June, 1954) discuss long-term cortisone therapy in rheumatoid arthritis. They state that 39 patients were treated who had suffered from rheumatoid disease for at least six months. None had crippling deformities or advanced joint destruction. An amount of 100 to 150 milligrammes of cortisone per day was given by mouth, and the dose gradually tapered down. Later, 75 to 100 milligrammes per day was the commencing dose, and a maintenance dose of 50 to 75 milligrammes daily was adopted after symptoms had subsided. It was not practicable to suppress symptoms completely. An average dose was given of 1·5 to 2·5 grammes per month. An initial course of three or four grammes was given with doses of 75 to 100 milligrammes daily. When this course ceased, later courses were given of 75 to 100 milligrammes per day up to 1·5 to 2·0 grammes. Gradual tapering off of the dose was preferable to sudden cessation. At this stage, 0·34 to 1·56 grammes per month were given. Intraarticular administration of hydrocortisone did not materially improve results. Some patients had intermittent and some continuous treatment. There were few cures. Most patients improved up to a point where they could undertake employment. Side effects noted were fluid retention in 33% of all cases, controlled by lowering the dose and restricting salt intake. Three patients had psychotic episodes. Degrees of moonface, acne, hirsuties and menstrual irregularities were noted, but were never a major problem. Thrombosis of deep calf veins occurred in two cases. It was concluded that prolonged treatment, either intermittent or continuous, was possible with cortisone provided that adequate supervision was carried out. Generally, results were rather disappointing.

"Largactil."

A. B. DOBKIN *et alii* (*Canad. M.A.J.*, June, 1954) discuss the uses of a new drug, "Largactil" or chlorpromazine, which belongs to the "Diparcol"-"Phenergan" series. It is said to depress the parasympathetic and sympathetic impulses, to be antihistaminic and to affect the temperature-regulating mechanism. For premedication in anæsthesia 150 milligrammes given by mouth overnight and 50 milligrammes given intramuscularly one hour before operation were found effective. In peripheral vascular disease, there was marked peripheral vasodilatation with a rise of temperature in the limbs. "Largactil" enhanced the value of aspirin, phenacetin, codeine and "Demerol" in relieving pain in inoperable cancer. This report is a preliminary one. Further studies are being made.

Heparin and Angina Pectoris.

K. W. G. BROWN AND H. E. RYKERT (*Canad. M.A.J.*, June, 1954) discuss the effect of treatment of *angina pectoris* by injections of heparin. They state that Lyon and others have described remarkable improvement in 93% of patients treated by this method. The authors treated 30 patients. Intramuscular injections of heparin, 10,000 units of a concentrated aqueous preparation, were given for three months, twice weekly. An equal number of patients was treated with an intramuscular injection of a placebo at the same intervals. All the patients, whether treated with heparin or a placebo, stated that their attacks of angina were less frequent during treatment. Heparin gave no better results than the placebo. No benefit was observed in seven patients with angina treated by intravenous injections of heparin; some ill effects were noted.

Advances in Management of the Nephrotic State.

J. A. LUETSCHER, Q. B. DEMING, B. B. JOHNSON AND C. F. PIEL (*J.A.M.A.*, December 5, 1953) discuss advances in the management of the nephrotic state. They submit observations made on the effects of treatment of 42 children and adults suffering with this condition. It is noted that bed rest stimulates diuresis in only the mildest cases, and it is without effect on the outcome of the disease. Also, in addition to its bad effect on morale, prolonged bed rest may cause undesirable shifts of fluid to the genitalia, abdomen or chest and may predispose the patient to venous thrombosis. A generous fluid intake is usually prescribed, but is not always accepted by the patient. Various special diets have been tried without convincing results. Restriction of sodium intake to less than one gramme of sodium chloride per day may be helpful, but this may also impair the total intake of food or may lead to an undesirable reduction of serum sodium concentration. Cation exchange resins have been of very limited assistance and have been poorly tolerated. The conventional diuretics are unreliable. Mercurial diuretics were not used because of the risk of serious immediate reactions and possible aggravation of the renal lesion. Concentrated human serum albumin induces a temporary diuresis in about one-half of the patients who receive 50 grammes intravenously for four or more days. Whole blood may be helpful in patients with severe anæmia. The control of pre-existing infections and the removal of foci of infection should be undertaken only in accordance with established principles of sound practice, not in the hope of alleviating the nephrotic state. Intercurrent infection is one of the chief causes of morbidity and death in nephrosis; the upper respiratory tract, the skin and the peritoneal cavity are especially vulnerable. Prompt treatment with antibiotics is essential for such infections. Continuous administration of antibiotics for prophylaxis has not been used except during treatment with cortisone or corticotropin. Cortisone, corticotropin and hydrocortisone were used in the authors' series of cases, and were given in full dosage over periods of ten to fourteen days' duration with abrupt withdrawal. In contrast to prolonged treatment, brief courses are safer and easier for all concerned and offer the double chance for improvement on either treatment or withdrawal. A daily dosage of less than 40 milligrammes of corticotropin or 100 milligrammes of cortisone is likely to be ineffectual, even in small children. Doses of more than 100 milligrammes of corticotropin or 300 milligrammes of cortisone per day are likely to be associated with pronounced side effects. The improvement that follows the use of corticotropin or cortisone in nephrosis varies from complete remission to no measurable effect. However, these hormones apparently shorten the nephrotic episode in certain cases. The lack of comparable controls makes it difficult to be certain whether the ultimate outcome in the whole group has been improved. One or more courses of treatment with moderate dosage of cortisone or corticotropin for two weeks reduced or eliminated symptoms and signs of the nephrotic state in a large proportion of cases. Repeated relapse or failure of treatment calls for some change in the plan of treatment. Larger dosage or more prolonged treatment with cortisone or corticotropin may be indicated in selected cases, but some increase in the hazards of treatment must be anticipated and controlled.

"Mysoline" and Epilepsy.

W. E. J. WILSON AND OLIVER HODGSON (*J. Ment. Sc.*, January, 1954) states that a small group of 11 epileptics have been treated with "Mysoline". Major epileptic activity was increased in eight cases out of nine. One patient was rendered free from fits, although his electroencephalogram did not improve. There was slight improvement in one-third of the patients with minor epilepsy. Electroencephalograms were

unimproved after administration of "Mysoline". "Mysoline" is therapeutically less effective than phenobarbitone or soluble phenytoin. In this series two patients developed *status epilepticus* during the transition from orthodox drugs. In one case this proved fatal. Of particular significance among the toxic symptoms were falls in the total white blood cell counts, which occurred in more than half the patients. This would seem to necessitate clinical examinations and pathological investigations greater than those required during treatment with established anticonvulsants.

Role of Antibiotics in Therapy of Acne.

H. M. ROBINSON (*Arch. Dermat. & Syph.*, April, 1954) states that the various antibiotic ointments were used in the local treatment of 72 patients with acne. A statistical survey indicated that such therapy of *acne vulgaris* with antibiotics produced temporary improvement in only a very small percentage of cases. The possibility of producing acquired contact sensitivity to these drugs by long-continued application is a major contraindication to such therapy. The author considers that broad spectrum antibiotics are of value as supplementary therapy in *acne vulgaris* and *acne conglobata* and do not replace X rays, dietary measures and proper local treatment. The local use of antibiotic therapy is not recommended. In the author's series, penicillin given by injection was of no value in the treatment of acne. Oxytetracycline, chlortetracycline and erythromycin were the drugs of first choice. Chloramphenicol is not recommended because of the possible production of blood dyscrasias. Long-continued administration of the antibiotics without rest periods is not advisable. Improvement may be maintained in a high percentage of cases by administration of subtherapeutic maintenance doses.

NEUROLOGY AND PSYCHIATRY.

Faith and Delusion in Psychotherapy.

JULES H. MASSERMAN (*Am. J. Psychiat.*, November, 1953) defines psychotherapy as being any procedure available to the ethical practitioner that helps the patient become happier, more creative and better adjusted to his family and society. He asks if it is possible, instead, that psychotherapy actually consists in the reestablishment of certain delusions necessary to all mankind. It has been his experience that there are at least three basic processes essential to man's "psychic" economy. He states that these three psychological manœuvres may be called the "Ur-defenses of man". Each of the three is contrary to the better evidence of our senses, and may be called "wishful", "fantastic" or indeed "delusional", and yet each is an article of faith believed by common consent. The first may be expressed as: "Nothing can really hurt me—I am inviolate." No man can conceive of his own death; he leaves "his successors", "his family", "his name" and "his will". The second is "the mastery of the omnipotent" and is designed to bolster and indemnify the "delusion of non-vulnerability" in a most subtle and satisfactory way. *There never was a man either so primitive or so emancipated as to lack a belief in some abstract being or agnostic principle or system. The third* (and to the cynic the ultimate delusion) is that in time of need one can seek *and actually obtain succour from one's* fellow man. Perhaps the "libido theory", a theory that traces all human *behaviour to narcissistic forms of* "eroticism", is an intellectualization of the third Ur-delusion. Unusual *introspective techniques must be employed* to sense the delusions with which man fills the terrifying vistas beyond the *limits of human understanding and* control. These delusions, expanded in scope of meaning, are necessary to *make each man's world seem a little* more like the heaven he wishes, and, in a deeply humanitarian sense, are indeed sacred. *Therapy must in fact be* directed towards restoring and respecting man's cherished and essential *delusions.*

The "Base Line" of Schizophrenia.

J. R. SMYTHIES (*Am. J. Psychiat.*, September, 1953) states that when the brain of the schizophrenic fails to work properly disorders of two types result: (i) disorders explained in mechanical terms, thought disorder, perceptual illusions and the disintegration between the environmental situation and the emotional response, characterized by this disease; (ii) disorders in which the normal psychic background becomes psychic foreground. Drawing from a collection of authorities who suggest that hallucinatory phenomena are of common occurrence in otherwise normal individuals, the author concludes that these phenomena appear when the activity of the brain is damped down through incipient sleep, fatigue, drugs or endogenous toxins, including the hypothetical toxins of schizophrenia. Because his brain has ceased to be able to suppress it, the schizophrenic sinks into contemplation of this inner world, which is said to have the splendours and transcendental beauty of the mescaline phenomena.

Dynamics and Classification of Disordered Behaviour.

SANDOR RADO (*Am. J. Psychiat.*, December, 1953) states that adaptation increases the organism's chances of survival and of cultural self-realization. The change may be in the organism itself or a change wrought by the organism on the environment. Adaptational psychodynamics are a development of classical psychodynamics. Behaviour disorders are disturbances of psychodynamic integration which affect the organism's adaptive life, utility and pleasure. The organized sequence of events is recognized as a process of miscarried prevention and miscarried repair. The classification is based mainly on the psychodynamic phases of ætiology, and overreactive, mood-cyclic, schizotypal, extractive, lesional and narcotic disorders are recognized. Dealing with overreactive disorders, the author states that overproduction of the basic emergency emotions of pain, fear and rage results in disorder. Failure to control overreactions results in emotional overflow into dreams and phobic, inhibitory, repressive and hypochondriacal behaviour. It is stated that in the schizotypes the psychodynamic integration is strikingly *inadequate; its organized action of* pleasure—its motivational strength—is defective, and expressed as "I do not *know how to react with people". The* zest for life is reduced, and sexual organization is rudimentary, ill-proportioned and subject to miscarried reparative patterns. When there is a lack of sustained and effective human relationship, *the response appears to be* rudimentary and rigidly limited to that of obedience or defiance. Three states *are recognized in the schizotypal* organization: (i) compensated schizoadaptation—the so-called schizoid personality; *(ii) decompensated schizo-*adaptation; (iii) disintegration and adaptive incompetence—the stage of *open schizophrenic psychosis.*

Effects of Aging on Cerebral Circulation and Metabolism.

P. SCHEINBERG, I. BLACKBURN, M. RICH AND M. SASLAW (*Arch. Neurol. & Psychiat.*, July, 1953) discuss the effects of aging on cerebral circulatory physiology. They chose 32 normal males between the ages of thirty-eight and seventy-nine years, and compared their data with those obtained from a study of 19 normal subjects aged from eighteen to thirty-six years. They deduce that there is a close correlation between advancing age and decreasing *cerebral blood flow and increasing* cerebral vascular resistance. The reduction in flow and increase in resistance are accelerated after middle age. Cerebral oxygen consumption remains fairly stable until after middle age, when it apparently begins to decrease with advancing age. The reduction in flow and increase in vascular resistance are believed to be due to increased cerebral vascular tone and sclerosis. Diminution in cerebral oxygen consumption is thought to result from impaired cellular function resulting from reduced blood flow. In eight of the cases, an inhalation of a mixture of 10% carbon dioxide and 90% oxygen resulted in apparent increases in cerebral blood flow of more than 100%. This indicates that the increased cerebral vascular resistance of the aged is not necessarily fixed.

Sclerosis of the Cornu Ammonis and Psychomotor Epilepsy.

K. SANO AND N. MALAMUD (*Arch. Neurol. & Psychiat.*, July, 1953) report their studies on the brains of 50 institutionalized epileptics who were studied with special reference to ictal psychic phenomena. In 29 of these cases they observed sclerosis in the hippocampal region and classified these cases into several groups according to their features. The incidence of emotional changes between attacks was high in these groups, and ictal psychic phenomena were present in 16 cases, the most prominent feature being rage. In 21 cases no sclerosis was observed in the *cornu ammonis*; in these cases the rage phenomenon was met with in only one instance, and ictal psychic phenomena and emotional changes were observed in only three cases. The authors state that a few electroencephalograms taken showed spikes or sharp wave foci mostly in the anterior temporal region on the side of sclerosis of the *cornu ammonis*. They discuss the possible role of the hippocampal formation in psychomotor epilepsy or temporal lobe automatism.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal Alexandra Hospital for Children, Camperdown, New South Wales, on June 17, 1954. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

Nutritional Anæmia.

DR. D. G. R. VICKERY showed a boy, aged eleven months, who had been suffering from scurvy with nutritional anæmia. He had been admitted to hospital in June, 1954, with the history that eight weeks previously he had developed painful limbs with varying amounts of swelling, and an odd posture with the knees lying outwards and the legs folded under him. Four weeks later, teething had caused swollen bleeding gums. The child was very irritable. He was found to be very pale, lying in the "pithed frog" position, crying bitterly when touched, and very apprehensive. He had a well-marked scorbutic rosary on the chest, his gums were bruised and bleeding, and his abdomen was softly distended with small hard mobile masses in the lower half. Both wrists were swollen, as were both his thighs, his right leg, and both ankles. The peripheral pulses were palpable, with a rate of 186 per minute. Jugular venous pressure was normal; the heart sounds had a tick-tack rhythm. The hæmoglobin value was 6·0 grammes *per centum*, and the reticulocyte count was 13%. Further examination the next day produced slightly lower values. The erythrocytes were very anisocytotic, but were mainly normal to microcytic in size. Central pallor was seen in some, and there were a number of polychromatic cells and poikilocytes. The neutrophile cells numbered 4410 per cubic millimetre, the lymphocytes 8060 per cubic millimetre, and nucleated red blood cells 1100 per cubic millimetre. Thrombocytes were numerous. The X-ray appearances of the chest and long bones were typical of scurvy. Only occasional red blood cells and leucocytes were found in the urine. A blood transfusion of 120 millimetres of blood was given, and treatment was begun with ascorbic acid; four doses of 500 milligrammes were given intramuscularly over three days, followed by 50 milligrammes twice daily by mouth. The diet consisted of whole boiled milk, "Farex", vegetables, orange juice and "Pentavite" with an iron mixture. He made good progress and after one week could be picked up without complaint, had better colour in his lips and cheeks and had lost most of the tense swelling in the lower limbs.

Acute Nephritis with Œdema.

Dr. Vickery's second patient, a child aged eighteen months, had been admitted to hospital four months previously with a history of swelling of the eyelids and insidious œdema for one week. The œdema was thought to be allergic in nature, but after full investigation proved to be due to acute nephritis. A full blood count two weeks after the child's admission to hospital revealed a moderate degree of anæmia, and this was considerably increased a further two months later. Dr. Vickery said that the child was presented to illustrate the occurrence of anæmia due to infection. The œdema was considerable at the time of the two blood investigations mentioned, and in view of the hydræmia it was thought that the actual anæmia might not be as severe as appeared.

Melæna.

Dr. Vickery then showed a baby who had been admitted to hospital on June 4, 1954, at the age of three days with a history of melæna occurring early that morning. The stool was normal in size and very dark; no bright blood was visible. Birth had occurred after prolonged labour, and the child had been taking breast feeding well. Although the child was pale, no abnormalities were found on examination of the cardio-vascular and other systems, and general nutrition was reasonably good. The hæmoglobin value was 11·4 grammes *per centum*, and the erythrocytes numbered 3,400,000 per cubic millimetre. The leucocytes numbered 12,000 per cubic millimetre. The stool was found to consist almost entirely of blood. The Coombs test result was negative, the child's blood was Rh-positive, the bleeding time was one minute, and the clotting time was three minutes. A transfusion of 80 millilitres of blood was given. Pyrexia occurring on the following day was treated with penicillin and streptomycin. Vitamin *K* was given daily for six days. By June 8, the melæna had ceased, and the child was taking feedings by tube. On June 9, a generalized patchy erythematous rash appeared, which was treated by suspension of penicillin treatment and application of a suitable local medicament. By June 11, the rash had disappeared and the child was progressing satisfactorily.

Portal Hypertension and Hypersplenism.

Dr. Vickery then showed a child who had previously been treated in the Royal Alexandra Hospital for Children in 1947 with pyelonephritis and hepatomegaly. At that time, the results of a blood count, red cell fragility test, glucose tolerance test and Wassermann and Kline tests had all been normal, and the urine contained no bile pigments, bile salts or urobilinogen. She subsequently remained well, but splenomegaly was found on a routine examination in 1953 and investigated at Prince Henry Hospital. There she was found to have a total erythrocyte count of 4,450,000 per cubic millimetre and a hæmoglobin value of 65%. The leucocytes totalled 1300 per cubic millimetre, 40% being neutrophile cells, 50% leucocytes, 7% monocytes and 3% eosinophile cells. The thrombocytes numbered 53,000 per cubic millimetre, and the reticulocyte count was 0·2%. The red cell fragility was normal; the bleeding time was three minutes, the coagulation time eleven minutes and the prothrombin time twenty-four seconds. The bone marrow was slightly hypoplastic. The results of liver function tests were normal. X-ray examination of the skull showed no abnormality, but there was a slight enlargement of the hilar shadows in the chest and rather loose trabeculation in the long bones. On further examination at the Royal Alexandra Hospital for Children, she was found to have a spleen enlarged to below the umbilicus; the liver was not enlarged but felt slightly nodular. The results of a blood count were much the same as before, excepting that the hæmoglobin value was 12·5 grammes *per centum* and the total leucocyte count was 2000 per cubic millimetre with a higher relative lymphocytosis. The red cell fragility in saline was normal. The urine contained increased urobilinogen. Liver function tests revealed a serum bilirubin content of less than 0·5 milligramme *per centum*, a total serum protein content of 5·9 grammes *per centum* (albumin 3·7 grammes, globulin 2·2 grammes *per centum*, albumin-globulin ratio 1·7), a thymol turbidity reading of 2·0 units, a positive result from the thymol flocculation test, and a serum alkaline phosphatase content of 46 King-Armstrong units. The blood urea content was 20 milligrammes *per centum*, and the Mantoux test result was positive with a dilution of one in 1000. X-ray examination showed no abnormality in the chest or long bones, but in the abdomen a large soft-tissue mass was visible in the left hypochondrium, with a small rounded shadow in the right upper quadrant. Examination of the bone marrow showed red and white cell precursors of normal numbers and appearances; megakaryocytes were increased in number, and some were immature. No abnormality was detected in an electrocardiogram. The blood group was O(IV) Rh-positive. A Coombs test result was negative. Splenectomy was carried out by Dr. T. Y. Nelson. The liver was finely cirrhotic, and there was portal hypertension, but little enlargement of the œsophageal vessels. The gall-bladder was grossly enlarged, but contained no stones. Histological examination revealed finely nodular hepatic cirrhosis with alterations in the spleen characteristic of portal hypertension. The child had since remained well, and her blood count was normal, but her liver was small, hard and finely nodular.

Subacute Leuchæmia.

Dr. Vickery then showed a boy, aged four and a half years, who had been first admitted to hospital in June, 1953, with a history of illness for three weeks consisting of fever and pain in the limbs, back and neck severe enough to limit movement. The fever had subsided in a few days, but the pains continued. No abnormality was found on examination except a basal systolic murmur. Salicylates were given. A blood count revealed a moderate degree of anæmia with leucopenia and relative neutropenia. The blood sedimentation rate was 60 millimetres in one hour (Westergren). X-ray examination of the chest and spine showed no abnormality. Hæmolytic streptococci were grown from a throat swab. After about a fortnight the child seemed well, and the blood sedimentation rate had fallen to normal, but after a further fortnight the temperature rose to 101° F., an apical systolic murmur appeared and the fauces were reddened. Penicillin was given as well as salicylates, but mild fever kept recurring. The blood sedimentation rate was then found to be 105 millimetres in one hour (Westergren). A blood count revealed that the degree of anæmia was somewhat less and the total number of leucocytes had increased, but there was an almost complete absence of neutrophile leucocytes and mild eosinophilia. Examination of the bone marrow showed that approximately 90% of the nucleated cells were immature blast cells; the red cell precursors were crowded out and relatively reduced by the large number of blast cells, and megakaryocytes were present in normal

numbers. A diagnosis of acute leuchæmia was made, and cortisone was given by mouth in a dosage of 200 milligrammes daily for seven days and diminishing doses for a further seven days. After one week, the proportion of neutrophile cells had returned to a practically normal value, though there was still some anæmia and leucopenia. By August 5, the child appeared perfectly well, and bone marrow biopsy showed complete remission of the leuchæmic process. He was discharged from hospital. A month later, a blood count showed 8250 leucocytes per cubic millimetre with no immature forms. On October 23 he developed fever, limb pains and restlessness, and his leucocyte count was 12,900 per cubic millimetre with 11% of immature forms. On November 6, the leucocyte count had increased to 14,600 per cubic millimetre, made up of neutrophile cells 19% and lymphocytes 80%, many of the latter being immature. He was readmitted to hospital, where he was found to have slight lymph gland enlargement and slight hepatomegaly with bruises on both legs. A further blood count on November 13 revealed a total leucocyte count of 20,000 per cubic millimetre, made up of neutrophile cells 4000, lymphocytes 6000 and blast cells 10,000 per cubic millimetre. A marrow biopsy showed that 70% of the cells were blast cells. X-ray examination of the long bones showed no abnormality. For this recurrence of his acute leuchæmic state he was again given treatment with cortisone and the blood count returned to normal. The bone marrow showed great improvement, but not complete remission. He returned home and remained well until December 4, when generalized limb pains recurred, and he was readmitted to hospital. On January 21, 1954, his leucocyte count was then 13,000 per cubic millimetre, made up of neutrophile cells 5850, leucocytes 2470 and blast cells 4680 per cubic millimetre. Marrow biopsy again showed a high proportion of blast cells. He was treated with mercaptopurine, and by March 5 his blood count had again become normal and the bone marrow showed improvement but not complete remission. He had since remained well, and his blood count was normal.

Lymphosarcoma.

Dr. Vickery's last patient was a child, aged twenty-one months, who in April, 1954, had had a feverish illness with sore throat, which subsided in about a week after treatment with "Chloromycetin Palmitate". Four days later the child was very pale and complained of sore gums, which bled. The temperature rose again, and the throat became very inflamed, and, as the child was still pale and irritable, it was admitted to the Wagga Hospital with a possible diagnosis of poliomyelitis. Lumbar puncture revealed the presence of five mononuclear cells per cubic millimetre in the cerebrospinal fluid. The hæmoglobin value was 5·5 grammes *per centum* and the leucocytes numbered 3500 per cubic millimetre, 30% being neutrophile cells, 64% lymphocytes and 6% monocytes. A blood transfusion of 300 millilitres of blood was given, and on the following day the hæmoglobin value had risen to 9·6 grammes *per centum*. The leucocyte total was 1800 per cubic millimetre. The child was admitted to the Royal Alexandra Hospital for Children on May 1, 1954. He was eating fairly well and taking fluids well and complained of a mildly sore throat. He was a pale, lethargic, sick-looking child with a fine macular rash all over the abdomen. His temperature was not raised. The liver and spleen were palpable below the costal margin, and there was mild to moderate generalized lymphadenopathy. Examination of the fauces revealed no abnormality. Bone marrow biopsy showed, instead of the normal 30% to 70% of polymorphonuclear leucocytes, only 4·2% with 32% of nuclear fragments, which appeared to be derived from the myeloid series. The pathologist commented that the nuclear remnants appeared to be due to degenerative changes in cells as a result of abnormal maturation, but that they might be due to leuchæmic changes. X-ray examination of the skull, long bones and pelvis showed no obvious lesion. A second bone marrow biopsy on May 13 showed an essentially normal picture without nuclear fragments. Penicillin treatment was commenced on the day of the patient's admission to hospital. The pathologist commented that the nuclear remnants had disappeared and had been replaced by normal myeloid elements and there was no defect in maturation. The hæmoglobin value was 12·5 grammes *per centum* and the leucocytes totalled 8000 per cubic millimetre, 68% being neutrophile cells, 31% lymphocytes and 1% eosinophile cells. The liver and spleen were a little more enlarged. The picture then remained much the same with considerable pallor until May 27, when a further marrow biopsy was taken. The pathologist stated that this specimen showed smear cells and degenerate nuclei similar to the first biopsy, but there were living cells present with a neoplastic appearance which could be leuchæmia. There was a corresponding depression of normal cells, especially of the myeloid series. No megakaryocytes were seen. The appearances were consistent with infiltration of bone marrow by lymphosarcomatous cells. Lymph gland biopsy was carried out. The gland was reported to be pale in its gross appearance. Microscopically the architecture was well maintained. The lymph cords were more densely cellular than normal. The cells were larger, and mitotic figures were more numerous than in normal hyperplasia. The pathologist said that he believed that the histology of the gland favoured a diagnosis of early lymphosarcoma, which was consistent with the as-yet aleuchæmic blood picture and atypical bone marrow state. This state of affairs continued until June 2, when abnormal cells were seen in the blood. Of a total of 4400 leucocytes per cubic millimetre, 9% were neutrophile cells, 68% lymphocytes, 3% monocytes, 10% eosinophile cells, 1% basophile cells, 3% metamyelocytes and 6% atypical lymphocytes. The leucocytes showed a shift to the left. The hæmoglobin value was 9·3 grammes *per centum*, and the nucleated red cells numbered 180 per cubic millimetre. Oral cortisone therapy was started, and the appetite improved and the weight increased. Subsequent blood counts revealed similar findings. One blast cell was seen in an examination on June 11. The patient's general condition continued much the same.

Tetanus.

DR. D. G. HAMILTON showed a boy, aged eleven years, who had been admitted to hospital on June 5, 1954, with a history that on May 18 he had run a stick into his nose. A pustule formed and the wound discharged. A piece of wood was removed from the wound five days before the child's admission to hospital. One week before his admission, the right side of the mouth was noticed to be drawn up, and two days before admission right facial paralysis developed. There was also difficulty in opening the mouth widely. On the day of admission the symptoms increased, and he had difficulty in eating, but no trouble in swallowing, and also difficulty in walking. On examination, the child had neck stiffness and flaccid right-sided lower motor neuron facial paralysis with twitching of the right side of the face. The mouth would open an inch, but was very rigid if further opening was attempted. The speech was slurred. The child was treated with antitetanic serum, 200,000 units in desensitizing doses, and then 100,000 units twelve-hourly for two days followed by 50,000 units for two days. Penicillin was given in a dosage of 1,000,000 units six-hourly as well as "Terramycin" suspension, 250 milligrammes four-hourly. Paraldehyde was given by a Ryle's tube and *per rectum*. During the first night, a number of spasms were not completely controlled with paraldehyde. On the following day he was examined by the tetanus committee, who recommended tracheotomy and drip therapy with glucose and "Scoline". With the patient under intravenous "Pentothal"-"Scoline" anæsthesia, administered by Dr. C. A. Sara, tracheotomy was performed by Dr. E. Goulston, and the wound in the nose was excised after local infiltration with antitetanic serum. Fifty thousand units of antitetanic serum were given intravenously, and drip therapy was commenced with one gramme of "Scoline" in 500 millilitres of 5% glucose solution. The pulse rate dropped and the colour improved after tracheotomy. The spasms were controlled with "Scoline" and light sedation was maintained with paraldehyde given by means of the Ryle's tube. Nursing procedures were carried out after sedation. The patient was nursed head down with frequent changes of position in a quietened, darkened room and tube-fed with eight ounces of protein milk four-hourly. The general condition was satisfactory, apart from an increased respiratory rate and considerable overbreathing. On June 7, the concentration of "Scoline" was increased to 0·3% to reduce the volume of fluid administered intravenously. He still had some spasms and trismus on stimulation, but they were easily controlled with "Scoline" and paraldehyde. On June 8, the child started to become more sensitive to "Scoline", and the concentration was reduced to 0·1%. On June 9 he had a sudden attack of cyanosis of unknown cause, but no apnœa. The general condition deteriorated greatly during the day, with a rise in temperature and pulse rate and poor pulse volume, slight cyanosis, poor respiratory expansion, cardiac dilatation and gallop rhythm. Administration of "Scoline" was ceased, and digoxin was given intravenously. He began to improve and continued to do so. On June 11 he regained consciousness, was cooperative and was able to swallow. He still had slight facial paralysis and some trismus and complained of pain in the jaw, but no generalized spasms. Improvement continued, and on June 15 the tracheotomy tube was removed. The nose would be repaired later.

Congenital Cardiac Defects.

DR. DOUGLAS STUCKEY presented three children illustrating various types of congenital cardiac defect. The first was a

boy, aged four years, with a ventricular septal defect. His history was that he was born after normal pregnancy and labour, but had always been a poor eater and had suffered from frequent colds and attacks of bronchitis. He had no shortness of breath on exertion, but a cardiac murmur was discovered at the age of five months. He was a thin child with a pulse of full volume and femoral pulsations. The jugular venous pressure was slightly raised, but of normal wave form. The left ventricular apex beat was diffuse. The right ventricular impulse was in the parasternal area. A thrill and systolic murmur (grade four) were detected, being maximal in the fourth left intercostal space at the sternal border. The electrocardiogram was within normal limits. The X-ray examination showed moderate enlargement of the heart shadow with a slight increase in the vascularization of the lungs.

The second patient was a boy, aged two and a half years, suffering from the tetralogy of Fallot. He also had been born after normal pregnancy and labour, but in the early months of life was noted to tire easily, and at the age of seven months developed cyanosis. He now became short of breath on exertion and lay down to rest after walking about 20 yards. His general nutrition was fair, but he had cyanosis and clubbing of the fingers. The cardiac impulse was right ventricular in type. A systolic murmur (grade three) was audible, being maximal in the second left intercostal space. The second heart sound was loud and single. His hæmoglobin value was 19 grammes *per centum*. An electrocardiogram showed marked right axis deviation and slight right ventricular preponderance; the electrocardiogram was typical of the tetralogy of Fallot. X-ray examination showed that the heart was not enlarged. The aorta lay on the left side. The pulmonary artery was not enlarged. The lung markings were, if anything, diminished. In an angiocardiogram, the aorta and pulmonary artery were seen to fill early and simultaneously. The findings suggested the tetralogy of Fallot with probable valvular stenosis.

The third patient was a girl, aged six and a half years, with patent *ductus arteriosus*. Born after normal pregnancy and labour, she had developed normally without dyspnœa on exertion, but tired easily. A murmur had been noted recently by the school doctor at routine examination. She was of fair nutrition with a normal pulse and a blood pressure of 115 millimetres of mercury, systolic, and 70 millimetres, diastolic. The jugular venous pressure was normal. Femoral pulsations were present. The cardiac impulse was left ventricular in type. A systolic thrill and a typical continuous murmur with late systolic intensification were detected, being maximal in the second left intercostal space in the mid-clavicular line. A short mid-diastolic murmur was audible at the mitral area. The second heart sound was normal, with splitting on inspiration. The hæmoglobin value was 13·8 grammes *per centum*. Electrocardiography revealed a slight left ventricular preponderance. X-ray examination revealed slight enlargement of the heart, a prominent trunk of the pulmonary artery and slightly increased vascular markings of the lungs.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

STRANGULATED HERNIA.[1]

[From the *Australian Medical Journal*, September 1, 1846.]

THE following cases are taken from the London Medical Times with the object of instituting a comparison between London and Colonial Surgery.

We possess the fundamental elements of usefulness and improvement in the course of instruction which we have all enjoyed; and which is more or less identified with each individual in proportion to the degree in which he loves his profession for its own sake, enabling him to remove disease, wherever his lot may be cast. Advantages may be found in the possession of the multiplied means which large towns in the Old World afford, but these advantages are overvalued: the multiplicity of these auxiliaries to surgical practice has the effect of perplexing the judgement, by shutting out the force and simplicity of truth: yet, we venture an opinion, that by reason of their multiplicity these inventions may be pernicious. Consider the cupidity of mankind who modifies the production of his art that he may excite the admiration and dip into the pocket of the young surgeon: and consider also the contemptible vanity of the operator who affixes his name to a knife or forceps: imposing, perhaps unwittingly, but not the less censurably, upon such as are ignorant of the surgery of bygone centuries. Barclay's pupils will doubtless remember his ludicrous exposure of such pitiable ambition, which even the ruins of Herculaneum have lately proclaimed and condemned. We say that Colonial Surgery must not be destroyed by the absence of these appliances, many of which are more ornamental than useful. For instance we prefer the phlebotomy lancet and a quill to the elegant Conteau caché invented for phymosis, and we consider a large ascites trocar preferable to the huge instrument made to puncture the bladder per rectum. In other words we must expect success from anatomical knowledge and think less of the ingenuity of inventors.[1]

[1] From the original in the Mitchell Library, Sydney.

[1] Three cases of strangulated hernia are quoted.

Correspondence.

THE COLLEGE OF GENERAL PRACTITIONERS: QUEENSLAND FACULTY.

SIR: Reference the letter from Dr. Adsett in your journal of the 16th instant regarding the College of General Practitioners, Queensland Faculty, will you kindly permit me to offer some remarks?

It seems to me that the criteria for membership are unnecessary and overdone. Surely the mere fact that one is a doctor should secure him a passport into the College if he is in general practice. What higher standard of ethics should he possess other than that necessary to keep him on the register?

Item (*c*) is difficult to understand. Surely post graduate training is equally desirable for a man with a busy general practice who finds no time to assimilate his journals as it is for the other man who has not practised for twenty years? Will you please tell us how we are to obtain two members of the Queensland Faculty to propose and second someone whom they have never met?

If the College of General Practitioners is to accomplish its purpose, it is certainly not finding the easiest way to do so by imposing extravagant and exclusive criteria.

I think most general practitioners would feel that only two desiderata should be satisfied, namely, (i) the applicant is on the register and (ii) he is also in general practice. As the College is being established for general practitioners, to deprive any of them of the right of membership would reduce it to a private or secret society. No one would like to see freemasonry grow up in the profession.

Yours, etc.,

B. J. BOUCHÉ, M.R.C.S., L.R.C.P.

District Hospital,
Boulia,
Queensland.
October 25, 1954.

EXTREME TACHYCARDIA FOLLOWING THE INTRAVENOUS INJECTION OF CHLORPROMAZINE.

SIR: We wish to report a case of extreme tachycardia following the intravenous injection of chlorpromazine.

The patient, a healthy man of nineteen years, suffering from otosclerosis, was brought to the operating theatre following premedication with 50 milligrammes of pethidine and one one-hundred-and-fiftieth of a grain of hyoscine. His pulse on arrival was 124 beats per minute, and his blood pressure 170 millimetres of mercury, systolic, and 100 millimetres, diastolic. Fifty milligrammes of chlorpromazine were given intravenously in two doses of 25 milligrammes. He was somewhat dulled mentally, but could answer coherently. His pulse accelerated to nearly 170 beats per minute. It was regular, but because of the high rate it was difficult to count with complete accuracy. The circulation appeared

otherwise to be satisfactory. His colour was good, and the systolic blood pressure was 140 millimetres of mercury. It was decided to proceed with the fenestration operation. Anæsthesia was induced with 0·35 gramme of thiopentone and 80 milligrammes of gallamine, and a number 10 tube was introduced into the trachea. Nitrous oxide and oxygen were given supplemented with additional pethidine and thiopentone. The pulse fell to 150 beats per minute and remained at that level, with a blood pressure of 140 millimetres of mercury. At the end of one and a half hours 0·5 milligramme of neostigmine was given intravenously. Ten minutes later the pulse rate had fallen to 112 beats per minute, with a blood pressure of 140 millimetres of mercury. The pulse rate rose to the pre-operative level (120 beats per minute) in one and a half hours.

Convalescence was uneventful with little nausea and no vomiting. A week later his pulse rate was 120 beats per minute, and his blood pressure 140 millimetres of mercury, systolic, and 90 millimetres, diastolic.

It would seem that this patient was one with a normally rapid heart action, whose response to chlorpromazine brought about extreme tachycardia. The response to neostigmine was marked.

Yours, etc.,

W. H. J. COLE,
Anæsthetist.
DENIS F. O'BRIEN,
Otorhinolaryngologist.

Melbourne,
October 20, 1954.

CAECOSTOMY.

SIR: Dr. E. S. R. Hughes, from an experience of 11 cases of acute intestinal obstruction due to carcinoma of the colon and rectum, advocates caecostomy as the procedure of choice in this surgical emergency (M. J. AUSTRALIA, October 16, 1954, page 617). I believe this is a retrograde step.

In my own series of 31 cases of acute obstruction of the large bowel from carcinoma of the colon (representing over half the total number of patients with carcinoma of the colon who have been under my care), the primary procedure has been: transverse colostomy in 22 cases, inguinal colostomy in three cases, lateral anastomosis of the ileum to transverse colon in three cases, caecostomy in three cases. Three of these patients for whom colostomy was performed died after this initial operation (all being frail, elderly and neglected), while a fourth patient died from pulmonary embolism. Of the 24 survivors (of colostomy or ileo-transverse anastomosis), four were left with a permanent colostomy and one with a palliative ileo-transverse anastomosis, the growth in each of these cases being judged inoperable. Resections were done in the remaining 19 cases, with one death (from post-operative volvulus of the ileum). About half the patients in this series were over seventy years of age, and one of ninety-three years made a good recovery.

I would reserve caecostomy as an operation of the last resort. Of the three cases in which it was performed in this series, two patients had obstructive growths at the hepatic flexure with such gross ballooning of the caecum that no other means of relief appeared possible. One of these patients died and was doubtless doomed to die, for the caecum was as large as a sugar bag and its contents as thick as porridge.

In the third case in which caecostomy was performed, a McBurney's incision was made on a mistaken diagnosis of appendicitis. Exploration through this incision (considerably more difficult than through a routine paramedian) revealed a tumour in the sigmoid colon. Although the patient survived, his condition gave rise to the gravest anxiety for about ten days after operation before he recovered from his obstruction, and I bitterly regretted the error which forced a caecostomy upon me. Subsequently, I resected the growth in the colon and later again performed a formal closure of his caecostomy which showed no inclination to close spontaneously. Although he is now very well, I would never again perform caecostomy if it were in any way avoidable. Nor would I regard a McBurney's incision as suitable for exploration. Even if the site of obstruction is accurately known (as from X-ray evidence), in most cases an adequate laparotomy is essential to ascertain, for example, that the obstruction is indeed due to carcinoma and not to diverticulitis of the colon.

It seems hard to believe that caecostomy has any advantages over colostomy in the relief of obstruction in these cases—that is, in dealing with the surgical emergency

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 16, 1954.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	4(3)	4(3)	6(4)	..	1(1)	..	..	..	15
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	1	..	1(1)	..	..	..	2
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	2(2)	..	..	..	..	..	..	2
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	13(13)	7(5)	23(22)	..	..	..	..	4	47
Diphtheria	6(6)	1(1)	..	1(1)	..	..	..	1	9
Dysentery (Bacillary)	..	6(6)	1(1)	..	..	..	..	..	7
Encephalitis	..	2(1)	..	..	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	1	..	..	..	..	..	..	..	1
Infective Hepatitis	55(25)	73(38)	..	..	5(4)	..	..	..	133
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1	..	..	..	..	..	1
Leptospirosis	..	..	7	..	..	..	..	..	7
Malaria	..	1	2(2)	..	..	..	..	..	3
Meningococcal Infection	4(3)	4(4)	1	..	..	..	..	..	9
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	7(4)	15(9)	10(1)	1(1)	2(1)	..	..	..	35
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	27(15)	..	..	33(26)	..	..	2	62
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	14(7)	29(18)	6(4)	..	2(1)	1(1)	..	..	52
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	6	..	..	..	6
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	18(11)	21(13)	4(2)	6(4)	9(7)	6(2)	..	..	64
Typhoid Fever	..	..	..	2(2)	..	..	..	..	2
Typhus (Flea-, Mite- and Tick-borne)	..	..	6	..	1	..	..	..	7
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

for which an urgent life-saving operation is necessary. It is surely a simpler and safer procedure to lift a loop of colon out over a glass rod than to deliver the loaded caecum and to suture its thinned-out coat to the peritoneum with all the attendant risks of perforation and peritoneal contamination. Even a colostomy in its early stages is a messy business for the patient and the nursing staff, but a caecostomy seems very much more so from my observations. Furthermore, I have never found the transverse colostomy to be any embarrassment in the subsequent performance of an adequate resection, while if resection is not advisable (as must occasionally be the case with an inoperable growth), then a colostomy is vastly more tolerable as a palliative than a caecostomy can ever be.

I trust, therefore, that until further evidence is available, Dr. Hughes's advocacy of caecostomy in these circumstances will be received with a critical reserve.

Yours, etc.,

85 Spring Street, Melbourne, October 25, 1954.

ROBERT S. LAWSON.

Reference.

LAWSON, R. S. (1953), "Carcinoma of the Colon and Caecum", *Alfred Hosp. Clin. Rep.*, 3:102. (This article refers to 25 of the cases mentioned above, while eight further cases have been under my care since it was written.)

Obituary.

GEORGE MACDONALD.

THE UNIVERSITY OF SYDNEY was fortunate in having the foundation of its Medical School well laid under three outstanding Edinburgh graduates—Anderson Stuart (physiology), J. T. Wilson (anatomy) and D. A. Welsh (pathology); all three were helped greatly by their chief laboratory assistants, John Shewan, Louis Schaeffer and George Macdonald respectively.

George Macdonald, the last survivor of the group, died on October 13, 1954. He came to Sydney about the same time as Professor Welsh in 1902, and was his right-hand laboratory assistant in the University department of pathology from then until 1935.

The part played by laboratory assistants or technicians in university and hospital departments is a most important one, and George Macdonald set a very high standard, which has been maintained by the many technicians trained by him.

Medical graduates of the University of Sydney from 1902 to 1935 will recall with appreciation the kindly assistance given them by "Mac" when they were passing through the department of pathology, because the students' welfare was ever in his mind.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 59 and 61, of September 30 and October 14, 1954.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Flying Officer (Temporary Flight Lieutenant) David Norman Bennett (039747) is transferred from the Reserve and appointed to a short-service commission, on probation for a period of twelve months, 29th July, 1954, with the rank of Flight Lieutenant.

Flight Lieutenant R. H. B. Sacks (036080) is granted the acting rank of Squadron Leader, 1st May, 1954.

Active Citizen Air Force.

Medical Branch.

Flight Lieutenant (Temporary Squadron Leader) D. A. S. Morgan, O.B.E. (04398) is granted the acting rank of Wing Commander, 15th September, 1954.—(Ex. Min. No. 90. Approved 5th October, 1954.)

Deaths.

THE following deaths have been announced:

BREARLEY.—Edwin Andrew Brearley, on October 29, 1954, at Sydney.

NELSON.—William Thomas Nelson, on October 28, 1954, at Sydney.

SEAGAR.—Edward Aitken Seagar, on October 28, 1954, at Sydney.

Diary for the Month.

Nov. 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
Nov. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 17.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 18.—Victorian Branch, B.M.A.: Executive of Branch Council.
Nov. 23.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 24.—Victorian Branch, B.M.A.: Branch Council.
Nov. 25.—New South Wales Branch, B.M.A.: Branch Meeting.
Nov. 26.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Tasmania: Part-time specialist appointments for the north-west coast of Tasmania.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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